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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

NOVEMBER 1948

PSYCHOLOGICAL PHENOMENA IN CARDIAC PATIENTS*

CARL BINGER

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WHEN my former teacher, the late Dr. Richard Cabot, retired from the faculty of the Harvard Medical School to become Professor of Social Ethics at Harvard College, he told me that he was going to keep his office on Marlboro Street for one purpose only and that was to reassure patients who had been informed by their private physicians that they had heart disease. He felt very strongly about the harm that many doctors unwittingly do by making a diagnosis of heart disease in the absence of sufficient evidence. Since those days, which corresponded to the end of World War I, we have learned a good deal about the coronary arteries and the nutrition of the heart muscle. As you know well enough our knowledge is still insufficient. We often cannot be certain whether or not the heart is structurally damaged. In the absence of such knowledge it is natural for us to be cautious. We need to protect not only our patients but, quite justifiably, our own professional reputations. If we warn a man never to run upstairs, never to walk into

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a strong wind, never to lose his temper, we may, to be sure, be protecting his coronary system from one standpoint, although we may be hurting it from another. I do not in the least mean to imply that we should not warn him so, but if we do, we are also almost certainly arousing anxiety in him and unless we protect him, as well as his heart muscle, against insults we are falling short of our obligations.

All this is an old story to you I am sure. I need hardly tell an audience of this sort that a large number of patients who consult their doctors because of complaints centering in the heart do not have heart disease; nor need I tell you that the mention of heart disease terrifies most laymen. It is part of your daily work to make differential diagnoses between symptoms resulting from structural alteration of the heart and those due to anxiety. You know very well that these two states often coexist—that anxiety and tension may increase breathlessness and lower the threshold of pain and of visceral sensations, just as pain and breathlessness may give rise to anxiety and tension. Those of you who have followed the recent studies of Dr. Harold Wolff and his associates[1] realize what we have all suspected, that even the rather trivial emotional disturbances of daily life alter the heart's function, raise the blood pressure and the cardiac rate, increase the stroke volume and, in general, decrease the circulatory efficiency of the heart.

Perhaps it should be pointed out that the symptoms of structural heart disease are remarkably constant from patient to patient. Once these symptoms have been mastered it is pretty safe to say that the other symptoms present are not due to heart disease, but to other causes. An accurate assessment of symptoms is the best means of differentiating between structural damage and cardiac disorders due to emotional disturbance. Such a differentiation is essential to intelligent handling of the cardiac patient, because the treatment in the two conditions may be quite opposed. But one must bear in mind that the patient with structural damage to his heart is, himself, often anxious, and emotionally disturbed and many of his complaints may be the result of this disturbance rather than of his cardiac lesion.

Pain and breathlessness are cardinal symptoms of heart damage but they are both often present in the so-called "cardiac neuroses." What can one say about the difference in character of these manifestations in the two states? Anginal pain, no matter where radiated, characteristically occurs in the mid-sternal line. Cardiac pain associated with anxiety

or tension is much more likely to occur over the apex. Unless a neurotic patient has been briefed by witnessing a true anginal attack he will very rarely complain of mid-line pain. Anginal pain characteristically lasts from one to two minutes and is very quickly relieved by nitroglycerine. Pain associated with anxiety or tension often lasts much longer and although it may be relieved by nitroglycerine, relief is slower in coming. The patient with true angina usually knows what brought on the attack of pain, whether it be due to exercise or excitement, and he will immediately stop doing the thing that caused his discomfort. The other patient usually does not know how to account for his pain—which may come on at any time, even while resting comfortably and while apparently relaxed. Furthermore, the patient with true angina has great difficulty in describing the quality of his pain while the other patient can cite chapter and verse about it.

There are analogous differences in the dyspnea experienced by the patient with structural heart disease and by the patient without. The first is more likely to have panting respirations, the second more likely to feel as if he couldn't take a deep breath. Again, orthopnea is more common in the first than in the second.

Other differences could be enumerated. But these facts are widely known to more enlightened internists. If that is so, what then has psychiatry to offer further to this subject? I think it has a not inconsiderable contribution to make to the management and therapy of your patients.

I should like to discuss with you, therefore, some of the psychological phenomena seen in cardiac patients. Since this subject is a large one and the time allotted to me is limited we shall make better headway by restricting the discussion to three aspects of the problem. These three aspects will also coincide roughly with three age groups. The first group consists of children and young people who suffer from various forms of rheumatic heart disease. The second group consists of people of early and middle age who suffer from so-called "cardiac neuroses" and the third group, which I shall deal with only briefly, consists of people of middle and late middle life who suffer from coronary insufficiency and coronary occlusion. This division into age groups is, of course, somewhat arbitrary but, even if not wholly accurate, it will serve our purpose best if we permit it to stand.

Concerning the first group, composed of youthful, rheumatic car-

diac invalids, I think it is probably accurate to say that their psychological difficulties are, in general, subject to several important variables (omitting for the moment the personal characteristics of the patient himself): One is the severity of the cardiac disability; another is its duration and still another is the nature of the medical management to which these patients are subjected and, perhaps most important of all, the attitude that their parents take toward them and their illness. Whether these patients are seriously disabled or not, certain psychological difficulties invariably arise, especially so if the illness confines them to inactivity or to close supervision for any protracted period.

Let us now consider the nature of these problems: What psychological phenomena are we likely to find? Are they confined to the patient himself? What implications can be gleaned from them as to therapeutic management?

Here is a case in point:

Johnny is the eldest child of three. He is just 13; a skinny, lanky, active, intelligent, good-humored boy. He is well liked at school, a reasonably good student and is very keen on basketball and wrestling. He plays forward on the Junior High School basketball team. He has been no special trouble to his parents and, except for an extremely disorderly room and a great disinclination to wash and bathe, they seldom have to correct him. But he has not gained weight and often seems listless and tired and has looked pale and has had dark circles under his eyes. In addition, he has complained of vague pains in his legs and recently had one or two nose bleeds. All this has caused his mother some concern.

One evening he complained of headache and felt feverish. He had no appetite. He slept poorly and when his mother went in to see him he complained that his legs ached. The next morning she sent for their pediatrician. There seemed no doubt at all about the diagnosis. Johnny's mother, who was well posted in medical matters, said after they had left the room: "What about his heart, Doctor? I saw you listening to it for a long time." The Doctor said: "He has a slightly suspicious murmur; we'll have to watch it and keep him perfectly still for the present." That was easy because Johnny felt too wretched to do anything but lie quietly in bed and take the medicine given him. In a week's time he was a good deal better. But the doctor continued to come every day and always listened carefully to the boy's heart. Johnny was fas-

cinated with the electrocardiogram and the doctor explained all about it to him. His mother stood at the bed-side with her eyes glued on the doctor's face. She was always cheerful and controlled but naturally she was terribly worried. The doctor explained to her that although the valvular damage might be slight, what counted was the heart muscle which needed protection while the process was active; that Johnny would have to stay in bed until there were no signs of activity and that when he returned to school he would still have to be careful. He added: "I guess there'll be no basketball this year." His mother knew that would be the greatest blow of all but she counted on the boy's common sense and coöperative spirit.

This story has a happy ending. The boy made a good recovery. But you can see he was fortunate. He had a careful doctor, an intelligent and understanding mother and he himself was sufficiently alert so that his energies could be diverted into channels other than basketball and wrestling. Partly through the interest which the electrocardiogram had awakened in him he set to work to assemble quite a respectable radio.

If we vary any of the ingredients of this story we might have quite a different situation on our hands. A less biddable youth than Johnny, for example, a somewhat unruly, defiant, rowdy adolescent at loggerheads with his parents may present a trying therapeutic challenge. This is especially true if the child feels the restrictions of his illness to be a kind of punishment or if the parents ease their own concern and hostility by exploiting the situation and threatening the child. Even trained nurses have been known to say to such children: "If you don't keep quiet the doctor says you're liable to drop dead of heart failure."

From a psychological point of view there are two principal problems to deal with: One is in the patient, the other is in his parents. The chief problem in the child is to prevent boredom and to still the restlessness which so quickly overtakes him when he is deprived of his accustomed bodily activities, and finally to teach him what he can and cannot do, depending on the amount of cardiac damage. Early in his illness the child can be lifted from his bed into a comfortable steamer chair. Such a change in posture puts no extra strain on his heart, but on the other hand, may improve his morale greatly.

Soon after the acute toxic symptoms have subsided the services of a visiting teacher are essential. The longer the child is sick the more necessary this becomes—not so much to prevent loss of schooling as to pro-

vide some interest and substitutive activity and to prevent his slipping into a regressed, withdrawn state in which his chief satisfactions are in day dreaming. If lessons can be combined with, or supplemented by, interesting occupational therapy so much the better.

The problem of the parent is the more difficult to deal with and the more important. The bugaboo of rheumatic fever is the ever-present danger of recurring attacks with further damage to the heart. It is difficult to instruct a mother to be on the lookout for vague symptoms of reinfection, which are the common ones in childhood, without arousing undue anxiety in her and eventually by contagion, in her child. If we do arouse too much anxiety then she will over-protect her child, worry over him, fuss over him, keep him in bed long hours, urge him to eat and be alarmed at the slightest headache or evidence of fatigue. Your own imagination or experience can describe how a spirited child or a complaint one will react to such treatment. Or again, an over-anxious mother may, because of her fear, be blind to fairly obvious signs of trouble and assume a casual attitude which may be even more damaging than the over-solicitous one.

I have no magic formula to apply to this puzzling problem. But this much I can say: If the doctor aims to provide an atmosphere in which recovery from the first attack is facilitated and recurrences are to be prevented, then he must address himself to the concerns, to the worries and to the emotions of the mother. It is probably almost as important to listen to her as it is to listen to the patient's heart.

Although I have emphasized the place of the child's mother in all of this, the attitude of physicians, of nurses and attendants—whether in a hospital, an out-patient department or a cardiac home, is often of determining importance. If the child is to recover from the body blow that nature has dealt him and is to regain a place in his world, no matter how handicapped, then he must be taught the two most difficult lessons of childhood, if not of life: one is to defer an immediate satisfaction for an ultimate gain; the other is to accept certain inexorable limitations of his capacities. He will learn these lessons best if those responsible for him are themselves not over-burdened with anxiety. We should not, of course, fail to recognize the anxieties that the child himself is coping with. These may be mobilized and aggravated by the illness—though by no means solely caused by it.

Throughout this paper you will hear the word "anxiety" frequently

used. It is the most important psychological manifestation in cardiac patients. In the group I have just discussed, anxiety is more to be reckoned with in the parent than in the sick person. In the next group—sufferers from what are called cardiac neuroses—anxiety is of cardinal importance in the patient himself and in the formation of his symptoms. The term "cardiac neurosis" is a loose one. It is a diagnostic catch-all to define those patients who have unpleasant, often frightening, sensations which they attribute to their hearts or, again, who exhibit what we like to call functional disturbances of their hearts, such as tachycardia, palpitations and even sometimes extra systoles.

When a consultant examines such a patient and uses the most modern scientific equipment and after great care can find no evidence of structural damage to the cardiovascular system then the patient, if he has the temerity and extravagance to cling to his symptoms, is said to be suffering from a "cardiac neurosis."

Within the past few years it has become increasingly customary to send such a patient to a psychiatrist. When he arrives (let us call him Jones) the first thing he will talk about is the fact that he is in a psychiatrist's office. He's a bit shame-faced about that. He says he's never done such a thing before—as if he had been caught stealing money from his grandmother. This is an old story to the psychiatrist who soon puts his patient at ease. The next thing that comes up is the fact that Jones thinks the doctor thinks all of his symptoms are imaginary, otherwise he wouldn't have been sent to see a psychiatrist. This too is disposed of by the psychiatrist's reassuring and sympathetic attention. He explains to Jones that no symptoms are imaginary—if they are actually experienced—and that no one ever suspected him of malingering. The rest of the hour then is spent on a list of complaints and symptoms.

The psychiatrist pays close attention to the way in which the patient tells his story. He doesn't ask him too many questions in the first visit. What he is trying to do is to make a diagnosis—a psychiatric diagnosis, because on that will depend the choice of treatment and, in part, the prognosis.

Let us examine very briefly a few Joneses. Jones I is a man in the early fifties. For the last few days he has felt strangely apprehensive. He is tremulous and his hands are cold and moist. He says he has been having dizzy spells. On closer inquiry he does not actually experience vertigo, but momentary states of confusion with feelings of breathless-

ness and oppression in the region of the diaphragm. He has had difficulties before for which he has consulted a neurologist and he is accustomed to a certain degree of tension and apprehensiveness. This time, however, they are much more intense and disturbing. He feels panicky and convinced that there is something seriously wrong with his heart.

What happened was this: A year ago he was examined by his company physician, an excellent and well-trained young internist. This internist noticed in the electrocardiogram evidence of a wide S bundle branch block that had been present over a period of two years, according to previous electrocardiographic evidence. He said nothing to the patient about it, assuring him that everything was normal. A year later a new company physician re-examined him and told him there was something unusual in his E.K.G. This disturbed the patient greatly and since the two company doctors had not agreed he consulted a cardiologist. The cardiologist examined him carefully and made light of the finding, explaining it to him (at least so the patient told me), as a "slight surge of the S wave." This, of course, meant nothing to the patient and, let me add, it meant equally little to me. The only difference was, it was the patient's heart and not mine, so it didn't frighten me as it did him. I inquired as to the real meaning of the electrocardiographic changes and then took great pains to explain things to the patient and to reassure him. He was much relieved to discover that he was suffering only from his old familiar neurotic difficulties and he was able then and there to give up his heart disease. This was a gratifying, if superficial, result. The man leads an active, useful and full life and there seemed at that time no justification for exploring further into his emotional problems. It is, as you well know, by no means always so easy to lay a cardiac ghost as it was in this case of anxiety neurosis.

Take Jones II, for example. He is 30 years old, an extremely intelligent statistician. He was sent to me by an excellent cardiologist because the patient could not be convinced that there was nothing seriously wrong with his heart. He complained of palpitations at night. He would awaken with his heart pounding. He had vague pains throughout his chest. He said that at college he got too much exercise and had "sprung his heart." He said he was conscious of his "heart beating all around the bush." He said: "I was scared into a perfect jelly and thought of taking up religion to die a good Christian death." He was told not to dance and he began to be very careful of his health. He had many other bodily

preoccupations and spent hours every day brooding over his aches and pains and over his damaged heart. He had a bizarre and extravagant way of describing his feelings. He compared himself to "an old tooth waiting to be filled," and said that he "felt as if his heart were rotting."

The manner in which he presented his complaints, the semi-jesting, jocular, inappropriate affect which accompanied his bizarre and disturbing thoughts, as well as his fixed hypochondriacal preoccupations, convinced me that this was a seriously disturbed man with certain features to his make up characteristic of schizophrenia. This diagnosis was supported by psychological tests made by another examiner who described the patient accurately as an "intellectually compensated latent schizophrenic."

In a patient of this sort there is no use in attempting to approach the cardiac complaint directly. It has all the fixity and stubbornness of certain paranoid delusions. One can reassure him from time to time but reassurances do not stick. The wisest course is to disregard the symptoms and to address ourselves to the whole life-situation and to try to keep the patient in contact with others and with reality. This man had a brilliant military record, achieving high rank. His gifted scientific mind was put to excellent use. He even achieved a marriage of sorts but it was not distinguished by any deep feeling.

Jones III presents quite a different picture: a different diagnosis, different treatment and a different outlook. In November, 1945, I received a letter from Dr. Blank who wrote: "I am referring Mr. L.W.K. to you for your valued opinion and care. He is a high strung man with hypochondriacal symptoms and a labile hypertension." There was enclosed in the letter an abstract of the history and physical findings. The important facts were these: He was 36 years old; his blood pressure, in the right arm, was 180/100, in the right leg, 190/105. The patient had been hospitalized and given intravenous sodium amytal. After injection his blood pressure fell to 140/82. The heart was apparently normal and renal functional tests were within normal limits. An intravenous pyelogram showed slight distention of the kidney pelves. The diagnosis was "Essential hypertension with a very prominent nervous factor in its origin."

This patient came willingly for psychiatric help. He was very much frightened—frightened that he would be permanently invalided because of his hypertension. He saw himself in a wheel-chair after a

stroke. He feared a sudden heart attack, or that he might have to undergo a sympathectomy. He had many of the symptoms common in hypertensives: headache, buzzing in the ears, occasional precordial oppression and heaviness in the left arm. Further inquiry revealed a long history of neurotic complaints. He said of himself that he had always been overconcerned with his health. For a long time his gastrointestinal tract was the chief focus of his discomfort and concern but his anxiety had now attached itself to his blood pressure.

In spite of his disabilities he was an able and effective young business executive. His father too suffered from hypertension. I concluded that everything should be done to prevent this man's still labile blood pressure from becoming fixed at a high level. After further preliminary psychological studies I recommended psychoanalysis.

Two years after receiving the letter from the referring physician I wrote him the following letter:

Mr. L.W.K. had his last session with me on November 6th. As you know, he has had to be away a good deal on business trips. He has just returned from one to the West without his usual emotional and physiological upsets. This trip required his making several speeches which he was able to do without stage fright or cold sweats. His family doctor took his blood pressure about two weeks ago and found it to be 120/70. I think he has finally abandoned a conviction that he has hypertension, but he is still very gun shy of the blood pressure cuff. I imagine that he always will be.

You will be interested to know that he saw me 135 times for approximately 50 minutes at each visit."

Let me review with you briefly what transpired during the psychoanalytical treatment. As is usual in such treatment, one begins with the immediate and disturbing facts of the patient's life—the ones which he naturally first chooses to talk about. In this case it was his health and his worries over his future and the support of his family. What would happen to them if he should become permanently invalided? Gradually we penetrated behind this outer coating of defense and found in him a frightened, puny, pimply youth whose father had walked out on his mother. His father had had terrible rages and yet he was to him the ideal of a strong and heroic figure whom he remembered as an officer in World War I. But he never came back to them. The boy was left alone with an adoring, but bitter and unhappy, mother.

They lived in a part of the States where to be divorced was a permanent disgrace for a woman. The patient felt this keenly, became prematurely ambitious and self-reliant. He led his class at school but remained physically weak. He looked admiringly and enviously at the football stalwarts. This notion of weakness clung to him and in spite of more than usual success in his professional and domestic life it seemed to load the dice against him. Behind his driving ambition, there was great insecurity, great lack of self-confidence. What he actually wanted was failure and illness and to be taken care of. It was the old story of an unconscious wish being experienced as a conscious fear. All this was revealed to the patient—not as an intellectual process—but as a personal experience through which he passed gradually, accompanied by many disturbing emotions, including rage. Although the end result seems favorable, time alone will tell whether what he gained will remain with him permanently.

I have presented this patient as another example of an anxiety neurosis. He resembles the first one—the man who was so frightened because of his S wave, and the second one who was afflicted with hypochondriacal preoccupations. And yet the treatment in all three was different and in each case was aimed at the life-situation and personality structure, rather than at the cardiovascular symptoms.

There are many patients who exhibit far less overt anxiety than these three did. For example, I have in mind a woman of 38 whose history was one of tachycardia for a period of about 15 years. Her basic heart rate was usually 130-140 per minute, at times reaching a level of over 200. At other times, however, her rate dropped to 90 or even 80 to the minute. She had consulted a great many doctors (some very distinguished ones) and her list of medicines included atropine, digitalis, mecholyl, phenobarbital and quinidine. She had also had pressure exerted on her eyeballs, vagi and carotid sinuses, all without relief. And yet the heart was perfectly normal to examination. Nothing was ever found but paroxysmal auricular tachycardia.

She was repeatedly advised to have a psychiatric consultation. She admitted having had a "nervous breakdown" before the trouble began, but she said that she was quite able to analyze her own emotional difficulties and she saw no necessity whatever for psychotherapy. She had rejected all conceivable reasons for being nervous or tense.

In a patient of this sort it is sometimes impossible to proceed

psychiatrically. The whole emotional conflict is in a sense acted out by the heart. This is a true organ neurosis. Her unconscious drives are so threatening to her that they cannot reach the level of consciousness. They appear to find escape through a specific visceral disorder. And yet, psychological tests made on this patient showed a neurotic disturbance reaching serious proportions.

It would take us a little too far afield to discuss how we arrive at our diagnostic formulations in these patients. Among other things, we take into account their attitude toward the disorder, whether symptoms are diffuse or circumscribed, the type of sense organ that is involved and whether the symptoms are subjective only or observable by others (Levine[2]).

Usually in these so-called "cardiac neuroses" we have to differentiate between hypochondriasis, anxiety neuroses, and organ neuroses, and sometimes hysteria, in its technical, not its popular sense. None is necessarily exclusive of the other but the indications for treatment may depend upon the weighting of these various components. In all of them anxiety plays the predominant dynamic role. I will not attempt, in this paper, to define anxiety but to emphasize only that it is not entirely equivalent to fear and legitimate worry. It is a complicated emotional state in which hostility or rage as a defense against fear is also mobilized. There are unconscious elements in anxiety that explain why it is not always accessible to a rational common sense approach, why the accompanying cardiac symptoms of anxiety so often persist in spite of our best efforts at reassurance. In any case, we need to know what to reassure about.

The psychiatrist must make up his mind whether to explore and to uncover the deeper sources of anxiety or whether to help his patient cover them up and let healing occur by repression. The choice will depend upon the age, adaptability, intelligence and strength of character of the patient and on his capacity and willingness to relinquish symptoms.

In the older age groups, about which I will say a few words now, effective psychotherapy is by no means ruled out, but it is often difficult, especially in the presence of extensive cerebral arteriosclerosis. The group I have in mind are the sufferers from anginal attacks, from coronary insufficiency and infarction and from beginning symptoms of failure. Even though these patients have outspoken structural damage

to their hearts one must not conclude that emotional influences play no role in their illness or in their recovery.

Any experienced clinician can cull from his memory instances in which a profound emotional upset was followed by what we call a coronary accident.

A former President of the New York Heart Association recently told me of two such episodes in his clinical experience.[3] The first concerned a man who heard the screeching of brakes and the crash of automobiles in front of his house. He dashed to the window only to see his own son's car in the wreck; whereupon he was seized with gripping substernal pain characteristic of a coronary occlusion.

The second story was about an elderly physician with hypertension. He began to complain of slight indigestion and, being somewhat hypochondriacal, he feared at once that he had a cancer of the stomach. But he did not seek medical advice. Instead he brooded over his illness and said to himself: "If I have lost weight, then I surely have a cancer." After days of tortursome worry he finally summoned up enough courage to step upon the scales, but before it registered, he suffered a sudden coronary occlusion and died shortly thereafter.

Such histories could be multiplied out of your experiences I am sure. Perhaps it is wise to draw no etiological inferences from them—to recognize them as remarkable coincidences and to realize that coincidences are themselves facts of nature which merit study. We do know that outbursts of rage or startle-reactions, as from fright or the hearing of bad news, are sometimes followed by sudden cardiac deaths.

We have less information about the influence of prolonged anxiety or states of tension on the coronary circulation. On this matter our suspicions are not yet based on proven physiological observations. Inferences may, however, be drawn from illuminating observations, many of them emanating from Harold Wolff's laboratory, on the peripheral and visceral circulation.[4] The vascular supply to the mucous membrane of the stomach (and also of the nose) alters with the emotional state of the subject; rage appears to be accompanied by engorgement of the lining of the stomach and sudden fear by blanching. Analogous changes occur in the bladder, the vagina and the large bowel. From Mittelmann's[5] experiments we have learned that finger temperature may drop as many as 25° F. when the subject is in a state of anxiety. And again, Homer Smith's[6] studies have demonstrated a reduction in

renal blood flow up to 40 per cent under similar conditions.

The two original experiments of Homer Smith have been corroborated and extended by Pfeiffer,[7] working in Wolff's laboratory. In eighteen patients suffering from arterial hypertension rises in both systolic and diastolic pressures were observed when relevant life-situations were touched upon in interviews with them. Accompanying these rises in pressure there was a renal vasoconstriction reflected by decreases in the renal plasma flow up to 25 per cent of the control levels. This sometimes outlasted the rise in systemic blood pressure. In some cases the peripheral resistance to renal blood flow increased by as much as 40 per cent. Wolff and Pfeiffer conclude that the kidney in persons with arterial hypertension exhibits an abnormal vascular pattern characterized by increased arteriolar tone which is further increased by threats or assaults to the organism—physical or psychological. Such vasoconstriction, if prolonged, may damage the kidney parenchyma and eventually the brain and the heart. These conclusions support the theoretical ones proposed by others and myself in a monograph on the personality in arterial hypertension.[8]

Finally, let me add, that there is evidence in the literature that cardiac pain occurring under circumstances of emotional stress is associated with electrocardiographic changes.[9] Artificially produced extra cardiac pain may be associated with similar changes.[10] Whether these are the result of a diminished blood supply to the heart muscle because of vasoconstriction or are due to an extra demand for oxygen because of increased work is not yet clear.

Aside from considerations of etiology, however, sound therapy will have to take into account the patient's emotional state. There are some for whom prolonged bed rest acts as an irritant and whom sedatives will not quiet unless they are given adequate psychotherapy. According to recent observations made on Dr. Wortis' service at Bellevue, similar preparation is necessary in some patients before digitalis can be effective.[11]

It is my impression that almost every one who suffers an infarction goes through a period of depression, sometimes quite severe. It may long outlast electrocardiographic evidences of damage to the heart and may delay restoration to even moderate functioning. But these depressed states usually yield in time to the faithful ministrations of the physician and especially to a regime of gradually increasing activity. They do not often require the special services of a psychiatrist. There

is no better psychotherapy for the cardiac invalid than to be permitted and encouraged to exercise when such advice is compatable with his cardiac reserve.

The heart and the precordial area are quickly responsive to emotional stimuli and, reciprocally, sensations coming from these structures are often felt and interpreted as emotions. The Greeks, you will recall, looked upon the midriff as the seat of the soul and not only as an aphrodisiac to which one applies suntan oil.

Perhaps the poets could write for us a real treatise on the irregularities of the heart—that extraordinarily acrobatic and versatile organ which leaps and bounds and jumps and turns over and sinks and rises and catches fire and freezes and is light as a feather or as heavy as lead. Finally it stops, and that is what we are all afraid of.

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THE ROLE OF SODIUM CHLORIDE IN THE MECHANISM AND TREATMENT OF CONGESTIVE HEART FAILURE*

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IT IS a truism that the processes of disease result to a large extent in only quantitative variations from the normal physiological equilibria. In this sense, the response of patients with congestive heart failure to addition or subtraction of salt from the diet is merely an exaggeration of the reaction in the normal subject. In 1904 Widal and Javal[1] described a diuresis, an excretion of 10 to 12 grams of salt and loss of weight of about 1.75 Kg within 2 or 3 days of instituting a low salt diet in normal subjects; and, conversely, an oliguria, retention of salt and return to the control weight on adding 10 or 15 grams of salt to the low salt diet. Recent refinements of these experiments by Lyons et al.[2] have demonstrated, in addition, significant downward or upward changes in plasma volume, venous pressure and extracellular fluid volume. Similarly, Grant and Reischsman[3] have produced latent edema and plethora for a few days in normal subjects by adding 20-30 grams of salt to a normal diet.

It is my purpose to consider some of the reasons for the quantitative difference in response to salt in congestive failure, with special emphasis on renal factors. The clinical experiments which I shall present are the work of my associates, R. Mokotoff, R. E. Weston, D. J. W. Escher, L. Hellman, M. Cherkasky and G. Ross and their technical, dietetic and nursing assistants. My own role has been purely one of a trophic and regulatory center, sensitive, I hope, to stimuli from the periphery.

Since the beginning of this century, it has been known that there are two major components in the mechanism of edema, whether of renal or cardiac origin. From Starling's[4] experiments, the dynamic bal-

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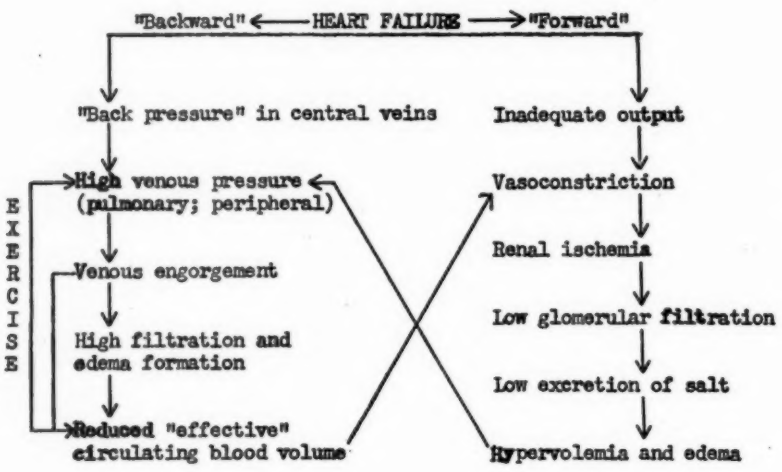
ance of physical factors in the filtration and reabsorption of fluid in the capillaries was clearly established. From Widal's[5] clinical studies, there was no doubt that retention of salt by the kidney was directly responsible for the degree of edema and gain in weight of nephritic patients. However, no simple combination of these two processes—the one, prerenal regulation of fluid exchange, and the other, renal excretion of sodium chloride and water—was integrated into the mechanism of cardiac edema, a basic feature of congestive heart failure. In fact, clinicians and their physiological colleagues soon became entangled in the foamy webs of colloid chemistry and the convenient mysteries of permeability, and succeeded in persuading themselves and others that the kidneys had little or nothing to do with renal or nephrotic edema and certainly little to do with cardiac edema. The major edemas were prerenally predestined and the renal component was largely a secondary matter of preserving a normal plasma electrolyte composition.

It was not realized for a long time that urinary output of salt alone could not be used as a measure of the causal relation of renal function to edema for the simple reason that there is a complex division of labor within the kidney itself which can influence the over-all figures enormously. For this job analysis of the kidney we are largely indebted to the ingenuity and industry of Homer W. Smith[6, 7] and his disciples. As a result of their far-reaching studies it is now apparent that the process of renal salt excretion must be dissected on a systematic basis to determine 1) the renal blood flow; 2) the amount of salt and water filtered in the glomeruli, which is the load presented to the proximal renal tubule, where the major or obligatory reabsorption of salt and water occurs; and 3) the residual load of salt and water presented for reabsorption to the distal renal tubule, where the final adjustments are made in the composition of the urine. When one considers that there are definite quantitative relationships between these kidney functions, that they are under the control of intrinsic renal and extrinsic nervous and hormonal factors, and that one of the chief functions of the human kidney is the conservation of base and water, necessitated by the enormous filtration of these substances in 24 hours, it becomes evident that no conclusion can be drawn regarding the role of renal salt excretion in the mechanism of congestive heart failure without careful quantitative study of renal hemodynamics in this condition. This approach was first made by Seymour et al.,[8] Warren and Stead,[9] Merrill,[10] Lauson, Bradley and

CHART I



Cournand[11] in their basic studies of cardiorenal interrelations in chronic congestive heart failure and acute shock.

I suppose it is not necessary to define congestive heart failure before this audience. Nor, I hope, will you take issue with me on the statement that when the heart becomes inefficient and fails it is like the old Roman god, Janus, facing both ways–forward and backward (Chart 1). The signs and symptoms of backward failure are more obvious, more striking, more readily measured clinically in terms of venous pressure, size of liver, pulmonary rales and edema; the signs and symptoms of forward failure, with the exception of acute peripheral circulatory collapse, are more subtle and difficult to measure because we are dealing with cardiac output and blood flow to various organs. As D. W. Richards[12] has aptly commented, in backward failure there is still forward flow but with back pressure in the lungs or systemic veins. That this increased pressure is partly compensatory and actually maintains cardiac output at a higher level than it would otherwise reach was early appreciated by Starr[13] and McMichael.[14] However, regardless of whether the cardiac output is low, normal or high in the presence of elevated venous pressure in congestive failure, it can safely be assumed that, prior to failure, that same heart was capable of a higher output because it was

TABLE I. RENAL HEMODYNAMICS IN CONGESTIVE HEART FAILURE

(Data from R. Mokotoff et al.)

Subject	*Renal Plasma Flow*	*Glomerular Filtration*	*Filtration Fraction*
	cc. per min.	*cc. per min.*	*per cent*
Congestive Failure	191.5±41.9	66.8±10.9	37.8±7.0
Control	627±74.5	103±10.0	16.6±1.5

more efficient. To put it another way, if a more normal heart were suddenly introduced into the circuit its output would be much higher than that of the insufficient heart even in high output failure. We can safely conclude that in congestive failure the output of the heart is inadequate for the body's requirements even at rest, and certainly during exercise or the body's requirements even at rest, and certainly during exercise or any other condition that increases metabolism and the load on the heart.

Heart failure is a form of circulatory stress. It is well to remember that whenever the circulation is exposed to stress, whether from sudden large hemorrhage, loss of circulating plasma into a traumatized area, or other types of shock or dehydration, all associated with an inadequate cardiac output, a redistribution of available blood flow occurs whereby the more vital organs are supplied at the expense of blood flow to skin, muscles, gastro-intestinal tract and kidneys.[15, 16, 17] Marked reduction of renal blood flow and glomerular filtration develops in these situations of acute stress. It is significant, therefore, that Merrill[10] and later Mokotoff[18] have shown these changes (Table I) to be regularly present in chronic congestive failure, regardless of the level of venous pressure or the temporary absence of edema as the result of treatment; but somehow an expression of an inadequate cardiac output relative to the body's requirement.

This markedly reduced renal blood flow is not simply the sequel of renal venous congestion, because it occurs even with only slight elevation in venous pressure. Furthermore, as the Bradleys[19] have shown in normal subjects, it takes a pressure of 14 to 22 mm. Hg in the renal vein—about 3 times the normal level—to produce a 25 per cent decrease in renal blood flow. Compare this with reductions of 50 to 80 per cent in renal blood flow in nearly all patients with congestive failure. A more striking difference between forward ischemia and backward con-

gestion in the kidney is seen in the glomerular filtration fraction. The Bradleys in their abdominal pressure experiments, found a proportional reduction in renal blood flow and glomerular filtration—hence, an unchanged filtration fraction. However, their experimental procedure not only increased post-glomerular resistance but also increased renal intrapelvic pressure, which could explain this deviation from the expected result. On the other hand, in congestive failure the glomerular filtration is never reduced as much as the renal blood flow; hence, the filtration fraction reaches abnormally high levels, an excellent compensation if you so wish to interpret it. Naturally, in a patient who has a high venous pressure and a tight abdomen full of ascitic fluid, the resulting renal venous congestion will add a further hindrance to the diminished renal inflow of blood and bring about a greater reduction in glomerular filtration of salt, beside producing a more severe oliguria. The dramatic improvement after abdominal paracentesis may well be the result of relief of renal venous congestion in such cases.

One of the important implications of reduced renal blood flow and glomerular filtration in patients with heart failure is the effect of exercise. The renal function measurements are usually made on patients at bed rest and under basal conditions. They are, therefore, probably maximal values since, as Merrill and Cargill[20] have shown, light exercise or ambulation regularly reduces renal blood flow by diversion of blood to muscles, and, in congestive failure, lowers glomerular filtration further. Also, exercise increases the venous pressure for relatively long periods in the cardiac patient, comparable with the findings of Landis et al.[21] that exercise increased central venous pressures in dogs with auricular fibrillation or ligation of a coronary artery. Therefore, just as in subjects with hunger edema, described by Denz,[22] the tendency to persistence or recurrence of edema will be much aggravated in the cardiac who is up and around. He will filter more fluid into his lower limbs and at the same time have a reduced urinary output of salt for which his kidneys may not be able to compensate during the hours of rest. The stage is therefore set for an attack of nocturnal pulmonary congestion since the failing heart cannot respond to the increased circulating volume during recumbency by adequate increase of cardiac output. The result is elevation of pulmonary arterial and capillary pressures. Other factors, of central nervous origin, are also involved in these attacks.

Another important point in renal hemodynamics has occasioned various interpretations and some misunderstanding. This concerns the role of reduced glomerular filtration in the retention of salt in congestive failure. It has been argued that the amount of salt excreted daily by the normal person is so tiny a fraction of the amount filtered that even a 50 per cent reduction of filtration should make little difference when one considers that a minute change in tubular reabsorption could compensate for it. Therefore, there has been a tendency for some investigators to disregard the lowered glomerular filtration in congestive failure, and, instead, to emphasize the possibility of increased tubular reabsorption of water and salt, presumably the effect of increased anti-diuretic hormone and other hormones. Much has been made of the lack of exact correspondence between the degree of reduction of glomerular filtration and the output of salt.[23] In all this type of argument it is sometimes forgotten that one of the chief functions of the mammalian kidney is conservation of salt. For this reason, as Homer W. Smith[24] has pointed out, the filtration rate becomes very important precisely because only a small fraction of filtered saline is marked for possible rejection. Therefore, salt is dealt with as a threshold substance, like glucose, but in a peculiarly different manner. Instead of having a maximum tubular reabsorption of so many milligrams per minute as in the case of glucose, there is probably only a maximum reabsorption, by the proximal convoluted tubule, of a certain amount of sodium for every 100 cc. of glomerular filtrate. This, of course, is necessary to maintain the isotonic concentration of sodium in the plasma and relates sodium to water reabsorption. However, the volume of glomerular filtrate which can be dealt with by the tubule in this manner is relatively unlimited, as shown by Pitts[25] and Lotspeich[26] for chloride and bicarbonate ions, the chief partners of sodium in the extracellular fluids. The kidney is therefore set primarily to regulate the concentration of salt in the body fluids and is relatively insensitive to the volume of extracellular fluids as long as they are isotonic. This evolutionary habit of the kidney is a major obstacle in the management of edema.

When glomerular filtration is reduced, the load of saline for the proximal tubule is correspondingly diminished and, finally, the distal tubule is presented with so little salt as to fall well below its maximal reabsorptive capacity, since only about 15 per cent of the glomerular filtrate normally reaches the distal tubule. Here water and salt reabsorp-

tion can be widely dissociated, thanks to the action of anti-diuretic and perhaps other hormones and acid-base regulation. The smaller load of salt may be practically entirely reabsorbed as part of the normal conserving activity of the distal tubule. The result is salt retention in the body and all the consequences that flow from it. You can see, however, that the initial and determining factor in this sequence is reduction of glomerular filtration, with upset of the normal glomerulo-proximal and proximal-distal tubular balances.

To use simple terminology from the economic sphere, we might consider glomerular filtration as a supply bureau for essential saline, of which normally 98 to 99 per cent is returned via the tubules for the domestic or internal economy while 1 or 2 per cent is routed for export into the urine. The distal tubule is the export control bureau. If supply or filtration of saline is excessive, because of brisk import of raw materials, ingested salt and water, export volume rises rapidly both in absolute units and as percentage of total filtration as a new equilibrium is established, with a somewhat increased internal supply. However, if glomerular supply or filtration falls toward the minimum required for the body's economy, as in extrarenal loss of body fluids or salt privation, export is not simply reduced proportionately but is largely or entirely cut off, as it should be in the interests of the internal economy. Unfortunately, in cardiac failure, when internal transportation becomes congested and is rerouted, the glomerular supply bureau receives less than the normal quota of saline although production of saline continues because of unrestricted import of raw materials. The proximal tubule being devoid of intelligence necessarily passes on a much smaller amount for export in spite of the fact that extracellular warehouses may be filled to the bursting point. The distal tubule, receiving a lesser volume of saline, reabsorbs nearly all of it. There apparently is no way by which the glomeruli or the tubules can be informed of the peripheral congestion and edema except by improving transport.

What is to be done under these circumstances? First, the external supply or import of raw materials, sodium and chloride, should be sharply reduced to prevent further accumulation of the finished product in the body; second, drastic measures must be taken to improve transport on the one hand and to relax export controls on the other. An intelligent physician will attempt to change the internal economy and to regulate the import of salt somewhat against the established habits or

individual liberties of the patient. The sooner this happens, the better.

Let us return to the evidence for the theory that the renal tubules are functioning normally in regard to salt in congestive failure. In our laboratory, Mokotoff et al.[18] have shown that the tubular reabsorption of sodium is set at the same constant level of 133 meq/liter of glomerular filtrate in the cardiac as in the normal subject on the same diet. This level is not affected by artificial increases in glomerular filtration. This figure, however, should not be taken too literally in estimating daily urinary excretion of sodium because it was determined with mannitol clearance as a measure of glomerular filtration. Mannitol unfortunately is an osmotic diuretic; therefore, it prevents the normal reabsorption of sodium somewhat and causes more salt to appear in the urine even in the cardiac patient with failure. Furthermore, slight changes in tubular reabsorption of sodium which might have a considerable bearing on the mechanism of cardiac edema would be very difficult to detect by the clearance methods. Whether studies with radioactive sodium will be more accurate in this respect has not yet been established. For the time being, it is a fair working hypothesis that the renal tubules are behaving normally in cardiac failure, that they are not under unusual hormonal influences, and that they respond in the usual manner to increases or decreases of the amount of filtrate and its content of sodium chloride.

An important question must be answered—what is the mechanism of the renal ischemia in congestive failure? Mokotoff and Ross[27] have investigated this problem. There are two possibilities—neurogenic or humoral. Using high spinal anesthesia to block autonomic reflexes and ephedrine to prevent fall in blood pressure, Mokotoff and Ross have found no significant changes in renal blood flow or glomerular filtration rate in patients with chronic congestive failure. These results would seem to exclude a neurogenic basis for the renal vasoconstriction. Merrill[10] has demonstrated the presence of renin in renal venous blood of cardiac patients in chronic failure. Whether this speaks for cause or effect of renal ischemia cannot be decided now. Other possibilities, such as Shorr and Zweifach's[28] VEM are being considered in searching for a humoral mechanism of renal origin. So far as the renal efferent constriction and the resulting high filtration fraction are concerned, they may stem from an intrinsic renal compensatory mechanism to counteract the effect of decreased blood flow on glomerular filtration pressure and filtration rate.

TABLE II. EFFECT OF ADRENOCORTICOTROPHIC HORMONE (ACTH) ON RENAL HEMODYNAMICS IN HYPOPITUITARISM

(Data from L. Hellman et al.)

Subject	*Period*	*Renal Plasma Flow*	*Glomerular Filtration*	*Filtration Fraction*
		cc. per min. per 1.73M²		*per cent*
F. S.	Control	362.5	58.9	16
(Male)	ACTH	599.0	100.0	16
I. S.	Control	483.0	80.2	16
(Female)	ACTH	784.0	125.7	16

To broaden the subject of possible mechanisms in control of salt excretion in heart failure, my colleagues Hellman, Weston and Escher[29] have approached the question of renal function from the endocrine aspects as distinct from the cardiodynamic viewpoint. It has been well established by the clinical studies in Addison's disease by Talbott et al., Waterhouse and Keutmann,[31] and by experiments on hypophysectomized or adrenalectomized dogs by White and his associates,[32, 33] that intact adrenal cortical function is essential not only for normal renal tubular activity but also for adequate renal blood flow and glomerular filtration. Since the adrenocorticotrophic hormone is the intermediary between the central nervous system and the anterior pituitary on the one hand, and the adrenal cortex on the other, we have administered this hormone to patients with anterior hypopituitarism and low renal blood flow and filtration rate. A striking increase (57 to 70 per cent) was produced in both these functions, as expected (Table II). The implications in congestive heart failure have not been exploited as yet, but we are considering the role of malnutrition in anterior pituitary function. We realize that glomerular and tubular effects of adrenal cortical hormone release may counteract each other. Thus, increase in filtration would favor clearance or excretion of salt and water, other things being equal. However, increase in salt-retaining adrenal steroids would enhance tubular reabsorption of salt. Just what happens in congestive heart failure under different experimental conditions must be the subject of further study.

When we think of the liver in connection with salt retention in

cardiac edema, two major possibilities arise—1) portal venous hypertension aggravating the effects of systemic venous congestion, especially in patients with long standing cardiac failure and enlargement of liver; 2) deficient inactivation of salt and water retaining hormones by a poorly nourished, congested, fatty or centrally necrotic liver. There are some facts available on portal hypertension; on the question of hormone inactivation, we are dealing largely with hypothesis and assumption. In extenuation it should be said that it is extremely difficult so to control conditions clinically or experimentally as to obtain clear proof of diminished inactivation of pituitary or steroid hormones by the liver in heart failure. Nor is it certain whether the increased antidiuretic substance excreted in the urine of patients with cirrhosis, as described by Ralli et al.,[34] is a primary or secondary event in relation to ascites and edema.

The discussion up to this point has indicated the central role of the kidneys in the mechanism of chronic congestive heart failure, or at least, of its most obvious signs, venous congestion and edema. We have seen that various hemodynamic and perhaps hormonal influences conspire to decrease the excretion of salt by the kidneys, which are innocent victims of the underlying circulatory disturbance. The diversion of blood from the ample renal blood supply to other regions of the body apparently is as constant a phenomenon in chronic congestive failure as in acute shock of various etiologies. A humoral mechanism is apparently involved in this redistribution of blood in chronic heart failure. The degree of the reduction in renal blood flow and glomerular filtration is more modest in the chronic cardiac than in severe acute shock, but its ultimate effect on excretion of salt is much more significant because of the time element. However, everything seems to point to the view that the kidneys are behaving as a conserving mechanism for salt in congestive failure, just as in the normal state or in conditions of salt depletion, in spite of the fact that there often is a large excess of saline fluid in the plasma and elsewhere in the extracellular spaces. Injected hypertonic salt solution, as Futcher and Schroeder[35] demonstrated so clearly, is eliminated at a rate less than 30 per cent of the normal in severe congestive failure, with consequent increase of weight due to edema. Excess of water alone, as Newburgh,[36] Schemm,[37] Schroeder[38] and many others have shown, is dealt with as in the normal by increased, although delayed, diuresis.

What practical conclusions can be drawn from the preceding physiological data to aid us in the management of congestive heart failure? It is a truism that little or no salt solution can be formed or accumulate in the absence of salt in the diet, no matter how potent edema producing factors are. The only exception is uremia, when even water may be retained and edema fluid of very abnormal electrolyte constitution result. There is no disagreement among clinicians as to the importance of restricting salt in the diet; there is a difference of opinion as to the degree of restriction and the practicability of the process. There is no problem in regard to acute congestive failure—any plan involving bed rest, digitalization, mercurial diuretics[39] and reasonable limitation of salt will usually be effective. The patient is ordinarily in a mood to coöperate and the results of treatment are soon apparent.

The situation is entirely different in chronic congestive failure. Here we face years of invalidism, the need for continuous medical treatment, the effects of drugs, of passive congestion and of psychologic factors upon the appetite and nutritional state of the patient. On the part of the physician, there is the philosophy of treatment. Should one insist on the diet with a minimal sodium intake compatible with freedom from edema or pulmonary congestion, or should one adopt the plan of a freer diet with frequent mercurial injections to hold edema in check? The latter is much easier for the doctor; the former requires considerable education of the patient and at least some knowledge of low sodium diets on the part of the physician. The situation is not unlike the present conflict in the management of diabetics who require insulin, with the significant difference that excess salt stays in the body while excess sugar runs out.

Let us agree at once that the cardiac patient who on casual limitation of salt requires only one or two mercurials a month is not really the subject of this argument. Let us confine our attention to the ever-growing number of patients who are receiving from one to seven mercurials a week, who spend most of their days travelling to doctors' offices or to clinics, who are completely useless economically and who, sooner or later, lose more and more of their own weight and respond less and less to diuretics. We have seen many such patients at the Montefiore Hospital react very well to a balanced low sodium diet, containing about 1.5 to 2.0 grams of salt. We have trained a fair number of these patients to continue on the diet at home. We have made it easier

TABLE III. EFFECT OF LOW SALT AND RICE DIETS ON RENAL HEMODYNAMICS IN HYPERTENSIVE PATIENTS

(From data of R. E. Weston et al)

Subject	*Diet*	*Renal Plasma Flow*	*Glomerular Filtration*	*Filtration Fraction*
		cc. per min.	*per 1.73M²*	*per cent*
G. B.	Full	393	104	27
	Low salt	372	88	24
	Rice	366	65	17
R. C.	Full	412	98	24
	Low salt	479	96	20
	Rice	364	66	18
	Full	491	101	21
A. M.	Full	223	54	24
	Low salt	238	37	16
	Rice	196	32	16
	Full	252	58	23

for them to take the most difficult hurdles in dietary management—the problem of bread and milk products. We have persuaded the nutrition department and our hospital administration to provide salt poor bread for distribution to out-patients. We have included sodium free milk ("Lonalac," Mead Johnson) in the diet, not to be taken unflavored as a drink, but to be used on cereals or in soups, custards, etc. Finally, we have made every effort to obviate the koshering or ritual salting of meat prior to cooking. The successful demonstration to even the mercury hardened patient that edema rapidly disappears and reaccumulates so slowly on our salt poor diet that only an occasional mercurial a month is necessary, has had a striking influence on the coöperation of the patient. Some have been restored to gainful occupation, most have been more comfortable than on any previous regime. Some are, of course, ineducable, just as in the case of some individuals with diabetes, obesity or peptic ulcer.

What are the disadvantages of the low salt diet? First, there is the change in flavor of food in spite of the liberal use of other condiments.

This may be very serious in some instances of malnutrition because there is no good substitute for salt in taste. On our diet, the permission of the use of 1 or even 2 grams of salt from a shaker, daily, may make a great improvement in the palatability of the diet without exceeding the renal excretory limit in some patients. Second, it is still very difficult in many parts of this medical metropolis to purchase salt poor bread and other necessary items of the diet, such as unsalted canned vegetables. Very few hospitals bake their own salt poor bread. Crackers, cookies, rolls, cake and other variants of the bakery are taboo for the unfortunate cardiac. What a field for an enterprising bakery! Third, there is evidence to indicate that a very rigid salt poor diet in itself may cause a further reduction in glomerular filtration and renal blood flow, something the very opposite of our therapeutic goals. Thus Weston, Hellman and Escher[40] have found as much as a 40 per cent reduction of the glomerular filtration in hypertensives without heart failure, on very low sodium diets like the rice diet of Kempner,[41] which, however, is also very low in protein (Table III). Mokotoff observed lesser changes on salt poor diets in normal subjects;[18] Perera and Blood[42] have recently reported similar results in uncomplicated hypertensives. It should be noted that these effects of low salt diets are not important in the usual case of congestive failure. However, they are of interest in the problem of so-called mercury-fastness or failure to respond to mercurial diuretics, a condition of importance in advanced stages of congestive failure. Let us examine this more closely.

It was stated earlier that the amount of salt excreted by the kidney involved a so-called threshold. Essentially, this means that the proximal and distal tubules separately exercise their reabsorptive capacities for sodium chloride and that what is allowed to escape into the final urine is simply the excess above these limits. This residual amount will be determined, therefore, by four factors—1) the serum sodium and chloride levels; 2) the glomerular filtration rate; 3) the maximal reabsorptive capacity of the proximal tubule; 4) the maximal reabsorptive capacity of the distal tubule.

Weston and Escher[43] have some evidence to show that the patient in severe congestive failure who is "mercury-fast" simply cannot filter enough salt to exceed the normal reabsorptive capacity of his tubules because of a combination of a low serum sodium and chloride level and a very low glomerular filtration rate. As a result, even the inhibiting

TABLE IV. EFFECT OF INCREASE IN GLOMERULAR FILTRATION ON RESPONSE TO MERCUXANTHIN IN "MERCURY-FAST" CARDIAC PATIENT

(From data of R. E. Weston and D. J. W. Escher)

Subject	*Period*	*Glomerular Filtration*	*Urine Volume*	*Serum Sodium*	*Filtered Sodium*	*Urine Sodium*
		cc/min.	*cc/min.*	*meq/l*	*meq/min.*	*meq/min.*
E. S.	Control	54.6	1.21	123.2	6.74	.002
	5 per cent NaCl	62.5	1.41	138.5	8.63	.004
	Mercuxanthin	83.5	12.50	135.2	11.30	1.222
		65.4	8.88	134.5	8.80	.725

action of mercury on tubular reabsorption fails to increase the urinary output of salt significantly. However, a sharp increase in glomerular filtration, produced by hypertonic saline or by aminophyllin, promptly converts the unresponsive to a responsive kidney (Table IV). The effect is temporary because of the difficulty of maintaining the increased filtration artificially without unpleasant side reactions.

Does one gain anything in edematous patients who happen to have low serum sodium and chloride, if one deliberately increases the salt intake in order to produce a better salt diuresis with mercury? The answer is "NO"! One may promote a higher excretion of salt during the mercurial diuresis, but the amount of administered salt is never eliminated entirely and the net result is a gain in the salt content of the body. As a matter of fact, the crux of this problem is the manner in which the extra salt is given. If it could be administered safely in very hypertonic form, and the fluid intake sharply restricted for some days to prevent formation of extra edema fluid, the resulting rise in serum sodium and chloride might ultimately alter the renal excretory pattern. Intravenous injection of hypertonic saline at the rate tolerated by patients in congestive failure seems to have little net effect on the urinary salt output in the absence of diuretic drugs. The data of Futcher and Schroeder[35] have clearly shown this, as well as the resultant increased edema in their cardiac patients.

What about the role of large quantities of fluid, as in the Schemm[37] regime, in the mobilization of edema? From the physiological experi-

ments of Wolf[44] on normal subjects on normal diets it is evident that the kidney cannot excrete urine with less than about 0.1 per cent of salt or 1 gram per liter. Gorham et al.[45] have applied this fact to edematous cardiac patients given a diet with only 2 grams of salt. They were able to double the small urinary output of sodium and reduce edema by raising the fluid intake from 1500 cc. to 3000 cc. or more. However, there was little difference in the total sodium excreted on 3000 cc. or 6000 cc. of fluids, probably because the kidney of the edematous cardiac can form urine with much less than 0.1 per cent of salt. Obviously, every extra gram of salt available in the diet will require one or more extra liters of fluid to be excreted, and under these circumstances the intake of fluid necessary to force loss of sodium from edema fluid over and above the sodium from the diet may easily reach 5 to 10 liters; amounts which, I am afraid, only thirsty Montanans could drink daily either orally or venously. Therefore, there is very little point to forcing fluids on the cardiac patient. There is also no reason for restriction of fluids. Perhaps we can agree on the elimination of the offending grams of salt from the diet by education rather than mercurialization.

As a final resort in the treatment of severe congestive failure when all the ordinary diuretic measures have proved inadequate, we suggest peritoneal dialysis with 5 per cent glucose solution in preference to frequent abdominal taps or the use of Southey tubes. Cherkasky and Hellman[46] have shown this procedure to be very effective and not unnecessarily risky. It entails primary electrolyte depletion of 10 to 20 per cent followed by secondary water loss, with considerable net loss of edema. The extra amount of salt removed from the peritoneal cavity in this manner corresponds to the output during an excellent mercurial diuresis. Adequate dietary restriction of salt will then help to maintain the patient at the new weight level or to slow up the rate of reaccumulation of ascites and edema. Poor results will occur in patients with severe renal insufficiency or in the moribund stage of congestive failure.

In this discussion, the role of salt in the mechanism of congestive heart failure has been presented from the renal aspect, although with no intention of neglecting the well-known dynamic exchange of fluids between the circulating blood and the extravascular tissue fluids. Emphasis has been laid on the compensatory renal ischemia in heart failure and the unfortunate effect on glomerular filtration of salt. The peculiar division of labor within the kidney makes it inevitable that reduced

glomerular filtration will always lead to retention of salt, hence edema and congestion, as long as tubular reabsorption is normal or increased. For this reason, treatment of congestive failure requires restriction of dietary salt, improvement of renal circulation and filtration by more efficient heart action, however produced, and inhibition of tubular reabsorption by mercurials or other means. The problem of the mercury-fast patient has been analyzed. The basic indication in chronic heart failure is continued and adequate restriction of salt in the diet. This involves an educational program directed toward physicians, dietitians, nurses and social workers, as well as to patients and their families; and should include an efficient mechanism for the wide distribution of salt poor bread and other essential dietary aids. The New York Heart Association might profitably add this attainable objective to its fine community service program as a goal for the immediate future.

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RECENT ADVANCES IN THE FIELD OF CARDIOVASCULAR DISEASE*

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IN considering recent advances in any field, one must first decide what is included in the term "recent." Since this decision was left entirely to the speaker, it has been decided to include chiefly events since 1940. The list as originally compiled was a long one, and it would be quite impossible to consider in the available time all the topics that seemed deserving of inclusion. It should be clearly understood that this is not a review of the recent literature; it is, rather, a personal selection from a long list of subjects that seemed to lend themselves best to the purposes of these lectures. It is quite certain that other lists, as long and representative as the following, could be prepared without difficulty.

The topics chosen for consideration fall readily into two main groups:—those which deal with diagnosis, including increased knowledge of etiologic factors, and those which represent progress in the prevention and treatment of disease. They will be discussed in that order.

Catheterization of the heart: Congenital anomalies of the heart have received a great deal of attention in the past several years, largely because of the successful correction of several of these by surgical means. One of the important diagnostic methods applied to the study of such lesions has been catheterization of the right side of the heart; a technique introduced many years ago, but perfected and brought into general use through the work of Cournand and his associates.[1] As most of you know, a small catheter is introduced into a vein of the arm, or the femoral vein, and under fluoroscopic guidance is passed steadily upward until it enters successively the vena cava, right auricle, right ventricle, and pulmonary artery. Samples of blood may be with-

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drawn as desired from the great veins and the right side of the heart for determination of its oxygen content, and the pressure can also be determined at different levels. If the oxygen content is significantly higher than normal in the blood from the right auricle or ventricle, one immediately suspects a shunt from the left side of the heart through an interauricular or interventricular septal defect. Analysis of blood from the right pulmonary artery may reveal a higher oxygen content than that in the upper right ventricle, which would suggest the presence of a patent ductus arteriosus pouring arterial blood into the pulmonary artery. The tip of the catheter can often be passed through septal defects into the left auricle or ventricle, as shown by fluoroscopic determination of its position and by the withdrawal of pure arterial blood. The safety of the procedure is sufficiently indicated by the fact that thousands of patients have now been subjected to it without serious discomfort and without injury. It is undoubtedly an important addition to our diagnostic methods and a technique that may yield even more important information in future with respect to fundamental problems related to cardiac physiology, function, and failure.

Neurocirculatory Asthenia: One of the significant advances in diagnosis in recent years has been that in our understanding of the condition known variously as irritable heart, disordered action of the heart, soldiers' heart, vasomotor instability, cardiac neurosis, DaCosta's syndrome, effort syndrome, anxiety neurosis, and neurocirculatory asthenia. It is my impression that greater progress has been made toward an understanding of this complex and serious condition in the past six years than in all the preceding decades since DaCosta first described it in 1871. A number of careful and thoughtful studies have been published in England and the United States, and while many of them are commendable, one cannot avoid particular mention of the splendid papers by Paul Wood[2] in England and those by the distinguished group at the Massachusetts General Hospital,[3] and by Friedman[4] in this country. Inasmuch as Wood's was the first of this recent group of fine studies, it is perhaps appropriate to quote briefly from it in order to indicate the trend of recent thought upon this subject.

In discussing the etiology of the condition he remarks: "We have seen that the symptoms and signs of DaCosta's syndrome are produced by central stimulation; there remains to be considered the origin of

the central stimulation. It must be emphasized that the condition is as common in civilians as in soldiers, and that even when present in soldiers, it was present before enlistment more often than not. It is clear, however, that trauma, high explosives, wounds, and gas poisoning are relatively unimportant. The chief factors seem to be the family history, constitutional incapacity, infection, neurosis, and undue mental or physical strain."

A detailed study of one hundred unselected cases immediately revealed two things: first, that a factor in the majority of cases was a fear of and dislike for Army life, and second that a psychiatric diagnosis could almost always be made—depressive state, anxiety neurosis, hysteria, hypochondriasis, etc. To quote again: "The essential common factor was the emotional reaction. The special feature which distinguished the group from other psychoneurotic physical patterns was the establishment of a link between the emotional reaction and effort. This link was usually forged when emotional symptoms were misinterpreted and the heart blamed, so that fear of effort followed."

His summary and conclusion are stated as follows: "1) The symptoms and signs of DaCosta's syndrome more closely resemble those of emotion, especially fear, than those of effort in the normal subject. 2) The mechanism of the somatic manifestations depends upon central stimulation, not upon hypersensitivity of the peripheral autonomic gear. 3) This central stimulation is emotional, and is commonly the result of fear. 4) The reaction becomes linked to effort by a variety of devices, which include misinterpretation of emotional symptoms, certain vicious circular patterns, the growth of a conviction that the heart is to blame, consequent fear of sudden death on exertion, conditioning, and hysteria. 5) Incapacity tends to be exaggerated consciously or subconsciously in order to protect the individual from further painful emotional experience. 6) It is urged that the diagnosis of "effort syndrome" be dropped. A proper psychiatric diagnosis is nearly always available; if attention is to be called to the presence of effort intolerance, let effort intolerance be added in brackets."

In that same year, 1941, there appeared in British journals many other studies[5] which while less extensive and conclusive than the one just quoted, are nevertheless important because the authors arrived independently at the same general conclusions, namely, that so-called effort syndrome is essentially of emotional origin and is closely related

to, if not identical with, anxiety neurosis. If we turn to our own country, we find that the Massachusetts General group have also arrived at practically the same conclusion, on the basis of extensive observations. Those of you who have read their fine series of reports, still incomplete, will recall that all recent papers have contained in the title the three terms "neurocirculatory asthenia, effort syndrome, or anxiety neurosis," used synonymously.

Friedman,[4] in a series of interesting studies, has also obtained apparently conclusive evidence that in this condition the symptoms arise as a result of a central stimulation which is emotional in origin. He has found that after mental or emotional rehabilitation, patients could perform exercise tests with normal cardiac and respiratory responses, whereas formerly these same exercises had caused marked disturbances in breathing. Moreover, in many patients there were spontaneous, sudden, inexplicable attacks of tachypnea or dyspnea occurring while at rest, accompanied by other evidences of sympathetic stimulation, indicating conclusively that these respiratory difficulties were of psychogenic or neurogenic origin.

It is difficult to believe that all these workers, using different groups of patients, in different countries, during war and peace, employing different techniques, could all make the identical mistake. On the contrary, the unanimity of opinion among recent workers is one of the strongest evidences that after three-quarters of a century, we can at last state with confidence that all the various names proposed for DaCosta's syndrome should be discarded, and the proper psychiatric diagnosis used in each case, despite the strong objections of the average patient to being labelled as a psychoneurotic. Proper treatment and eventual cure of the condition will never be achieved so long as we continue to regard it as merely an abnormal response to effort or a functional abnormality of the heart or vasomotor system. It is my hope that many of you will be so dissatisfied with this very inadequate summary of the recent evidence that you will read at least several of the papers to which I have referred so briefly.

Rubella in Pregnancy: For an important contribution to our knowledge of etiologic factors in heart disease we are indebted to Australian physicians, especially to Gregg[6] and Swan.[7] Several years ago Gregg made the startling observation that congenital cataracts in infants were associated in a high percentage of cases with the occurrence of German

measles in the mother during the early months of pregnancy. Further studies in Australia, New Zealand, England, and this country[8] have not only confirmed his original observations, but have also disclosed that rubella during the first few months of pregnancy is very likely to cause congenital anomalies of the heart, deafmutism, and other congenital abnormalities. Last year Swan and Tostevin[9] reported a group of 56 mothers who had some form of infectious disease during pregnancy; 40 of these had rubella, the other 16 had measles, chicken-pox, mumps, herpes zoster, or scarlet fever. Of the 40 cases associated with rubella, 36 infants had congenital abnormalities, and 19 of these had evidence of heart disease. The authors emphasize that in a number of cases the heart presented no definite signs of disease on physical examination, but the organ was enlarged as shown by a standard six-foot film; they regarded the diagnosis as doubtful in 5 of the 19. In 15 of the 19 cases diagnosed as heart disease, the rubella occurred during the first 3 months of pregnancy; in the other 4 at 3½, 4, 4½ and 7 months.

A review of the literature pertaining to this dramatic discovery was published last year by Aycock and Ingalls.[10] This indicated beyond reasonable doubt that rubella during the early months of pregnancy is associated in many cases with abnormalities in the infant. One cannot say that rubella always causes such abnormalities, or that it is the sole cause of them, but it is now possible to state with confidence that a baby is very likely to be deformed at birth if the mother acquires this disease early in her pregnancy. One hundred cases of such congenital abnormalities reported by eight different authors were analyzed by Aycock and Ingalls, who found that the rubella occurred during the first month in 30 cases, in the second month in 42, in the third month in 22, in the fourth month in 5, and in the sixth month in 1.

It is unnecessary to stress the great value of this knowledge, but perhaps it is well to remind you of the further implications of the discovery. It is immediately apparent that other virus diseases may also have a similar effect; indeed, in the report of Swan and Tostevin there were nine abnormal babies born of the sixteen mothers who had measles, mumps, herpes zoster, or chicken-pox during pregnancy. Studies upon this point are now going forward, which may lead to clearer understanding of this serious problem, and possibly to prevention of a large proportion of congenital malformations.

It is also clear that important sociological problems have been created by this recent knowledge, for the question of therapeutic abortion inevitably arises. The review by Aycock and Ingalls concludes with these words:

"Many authors have gone as far as to suggest that the justification for therapeutic abortion, if rubella (or other exanthemata) be contracted in the first two months of pregnancy, should be debated. Others have gone as far as to accept the available evidence as sufficient grounds for termination of the pregnancy. Such a far-reaching question can be approached with wisdom only when there are adequate statistical studies to establish the specific risks of infection at all stages of pregnancy. Knowledge from such studies would have to be interpreted not only in terms of actual risk of congenital anomalies, but as well in terms of the 'health of the mother' in continuing a pregnancy with such a known risk, and finally in terms of an informed public opinion."

Sodium: The introduction of radio-active isotopes, while still very recent, may result in significant advances in diagnostic and therapeutic methods in the near future. Most of you know that radio-active potassium has been used for several years in the treatment of polycythemia vera and skin cancer, and that radio-active iodine is now being employed in the treatment of diseases of the thyroid gland. An example of the possible diagnostic use of such isotopes has been provided by Dr. George Burch and his associates, whose beautiful exhibit dealing with the fate of sodium in the body must have been seen by many of you at the last Annual Session of the American Medical Association. Dr. Burch has graciously sent me the manuscripts of two unpublished papers which summarize much of the material displayed in that exhibit. It is these papers which form the basis for the following brief remarks.

Observations were made upon the relationship between weight, venous pressure, and the excretion of sodium in a normal subject, a patient recovering from congestive heart failure, and one whose heart failure was steadily increasing in spite of treatment.[11] The sodium excretion was followed by using radio-active sodium 22, which has a relatively long half-life of three years. It was found in general that the patients with congestive heart failure excreted less than normal amounts of sodium and water during the periods when heart failure was stationary or advancing, but greater amounts than normal during periods of improvement. The changes in weight and in venous pressure

were concordant—that is, they rose or fell practically simultaneously—but varied inversely with the excretion of sodium and water. However, a change in venous pressure or weight or sodium excretion could not be shown to precede a change in either of the other two. The normal subject had excreted 69 per cent of the injected sodium in sixty days, the patient who was improving had excreted 64 per cent, while the patient with advancing heart failure had excreted only 42 per cent at the end of two months. The findings were in accord with the generally accepted ideas of congestive heart failure, that is, if failure is present or increasing, the weight and venous pressure increase and there is an abnormal retention of sodium and water; during recovery the reverse is true. But Burch and his coworkers are careful to emphasize that the mechanisms concerned with retention of sodium and water in this condition remain unknown.

In a related study, Burch and his collaborators[12] used Na 24, which has a half-life of only 14.8 hours, to study the rate of diffusion of sodium through the vascular wall and its rate of excretion into the urine and sweat. Normal subjects and patients with congestive heart failure were used, and the sodium was administered by a single intravenous injection. It was found that sodium diffuses through the vascular wall at two rates, rapid and slow. In a normal subject the average rate of diffusion indicated that about 32 per cent of the total plasma sodium diffused out of the blood every minute. This approaches a diffusion across the vascular wall equal to about 18 pounds of sodium or 50 pounds of sodium chloride per day. The patients with congestive heart failure showed a more rapid rate of sodium diffusion from the vascular bed than did the normal subjects. These rapid rates of diffusion between the blood and the interstitial fluid, and the huge quantities of sodium involved, make it necessary to reconsider the role of sodium in edema formation. It is clear that relatively slight changes in the rate of sodium diffusion from the blood, and a disturbance in its excretion through the kidneys, might result in rapid and marked edema formation.

These papers from the New Orleans group are in the nature of preliminary reports but are of such fundamental importance that they must be extended. It seems probable that this new method may hasten our final complete understanding of the complex and very serious problem presented by fluid retention in congestive heart failure.

Surgery of Congenital Anomalies: It may seem to many of you a

waste of time to refer to the surgical treatment of congenital anomalies of the heart and great vessels, but no survey of the past few years can possibly omit this dramatic contribution. Indeed, it is so dramatic and has been so widely publicized in both the medical and lay press that there are probably few physicians who are not familiar with the broad features of the three operative procedures associated respectively with the names of Gross of Boston, Crafoord of Stockholm, and Blalock and Taussig of Baltimore. It has been only a short time since Gross first successfully ligated a patent ductus arteriosus, but there have been hundreds of patients subjected to this operation since he first demonstrated that it could be performed. As of October 1, 1941, he has himself performed either ligation or resection of the duct in 225 cases. In a letter to me of that date he says: "To date, I have operated upon 225 ductus cases. Forty-three were operated upon by some form of ligation—a procedure which I have now completely abandoned. In the last 182 patients the complete division of the ductus has been performed. I have never lost a patient during or after operation from the division technique itself. In the 182 cases with division there have been four deaths from various causes (one from staphylococcus mediastinitis, one from faulty anesthesia, two from cardiac failure). This gives a mortality rate of 2.2 per cent. This series obviously includes many patients who were excellent risks; it also includes a considerable number of patients who were exceedingly poor subjects for surgery. I feel that the operative risk in our experience for children is almost zero."

Most authorities are now agreed that such operation should be performed in almost every instance if the diagnosis is certain, since it apparently prevents the chief complications often associated with this condition, namely, stunting of growth, enlargement of the heart leading to congestive heart failure, and subacute bacterial endarteritis.

Secondly, it has been shown in the past several years that it is possible to eliminate the constricted portion of the aorta in patients who have the adult type of coarctation. In October 1944, Crafoord performed this operation successfully on two patients. Before these cases were reported in 1945 by Crafoord and Nylin,[13] Gross had already performed successful extirpation of experimentally constricted aortae in dogs, and shortly after Crafoord's brilliant demonstration, Gross also operated successfully in human beings. On September 30, 1947 he wrote me as follows:

"In the coarctation work, I have operated upon thirty patients. I have backed out of four of these because it seemed that the anomaly was such that it could not be corrected by surgical means. In the twenty-six patients from whom the coarctation was removed there were four deaths from various causes. In the surviving twenty-two patients I have had one who had no relief from hypertension but in the remaining twenty-one subjects there has been an excellent reduction of the arterial pressure in the upper part of the body. In general, these operations have been quite satisfactory in young subjects—from seven or eight years up to about eighteen years of age. However, they are exceedingly difficult beyond this age, and there are disheartening returns, as far as satisfactory results are concerned, in older subjects (due largely to the sclerosis which occurs so early in the aorta of these patients)."

In point of time, the latest of the three dramatic surgical procedures applied to congenital malformations was that devised by Drs. Taussig and Blalock for the purpose of correcting at least some of the more serious features associated with the tetralogy of Fallot. The one important principle underlying this procedure is to supply additional blood to the lungs by suturing a normal artery (usually the subclavian) to one of the pulmonary arteries. In the great majority of cases this results in remarkable improvement in the condition of the patient.

It would be impossible to overestimate the value of these new procedures in improving or entirely curing several serious conditions of the heart and great vessels. I wish to join all other physicians in voicing gratitude for these new advances in a field where for generations treatment was regarded as impossible. It is not with any desire to minimize these great achievements that I feel it necessary to remind you that the total number of patients with these three conditions is very small in comparison with the number of those who have the acquired forms of heart disease. We cannot rest upon these laurels, but must still seek measures that will diminish or abolish hypertension, rheumatic fever, and coronary atherosclerosis.

Low Sodium Diet and Unrestricted Fluids in Congestive Heart Failure: In my judgment, one of the very important advances of recent years in the realm of therapy is the steadily widening recognition of the importance of low sodium, high fluids, and an acid reaction of the diet in the treatment of congestive heart failure with edema. In 1941

Schroeder[14] showed that the restriction of salt to an amount below that excreted in the urine always resulted in a decrease of edema. When salt was restricted to amounts less than 1.0 Gm. per day, restriction of fluids was unnecessary; indeed, he noted that diuresis sometimes occurred when the patient was taking fluids liberally, and that it might cease when fluids were restricted. He concluded that restriction of salt was important in treating the edema of congestive heart failure, but that restriction of fluids was of little value.

Long before Schroeder reported this significant study, Schemm had been following a plan of treatment which recognized the importance of restricting sodium, but went much farther. The fundamental principles underlying this will not be discussed in detail, as they are clearly and logically presented in Schemm's three important papers.[15] Briefly, as enunciated by him, these are: 1) Each liter of edema fluid contains about 10 grams of an alkaline mixture of sodium salts and 1000 cc. of water. This edema fluid is subject to the same vicissitudes as the normal interstitial fluid; some of the water may be given up for vaporization, and the edema fluid consequently become more concentrated; in these circumstances, the patient may be severely dehydrated, even in the presence of massive edema. 2) The alkaline edema fluid remains in the tissues indefinitely unless the bicarbonate fraction of its sodium salts is used up by the constantly forming metabolic acids or by ingested acids. Acidification incites the kidneys to eliminate neutral or acid sodium salts; the water in which they were dissolved may be eliminated through the kidneys (diuresis) or may remain within the body to correct body fluid concentration and cellular dehydration, thus causing a disappearance of edema without weight loss and without diuresis. 3) Water reaches the kidneys for the formation of urine only after all the other needs of the body for water have been met; therefore, enough water must be administered by mouth or parenterally to meet these other bodily needs and supply an excess for the excretion of sodium. In most cases this means quantities of 3 to 6 liters of water per day. On the basis of these concepts, Schemm recommended a regime which would: 1) decrease the ingestion of the material essential for the formation of edema, and encourage the mobilization of the sodium already retained, by providing a diet very low in sodium and yielding a neutral or acid ash; 2) increase the normal effect of the metabolic acids by the administration of small amounts of acid drugs; and 3) facilitate the elimination

of the mobilized sodium through the kidneys and avoid the development of cellular dehydration by administering plain water in amounts that are adequate according to the established principles of water-balance.

It is perhaps not surprising that doctors in general have been slow in accepting this important contribution nor that today one frequently encounters physicians who have not taken the trouble to ascertain why it is logical to force fluids in the presence of edema. If they have the courage to try this form of treatment in spite of misgivings, they usually stop it abruptly if the patient gains weight or has a slight increase in symptoms during the first one or two days. But this is not a new and untried method; Schemm's first report in 1942 was based on the analysis of approximately 600 periods of treatment in 400 patients, of whom 375 had serious heart disease and more than 200 had gross edema. So far as one may judge by published reports, this treatment was largely ignored for several years, but recently has been attracting wide and favorable attention. Of the reports now available, brief mention will be made of only two. Leevy and his co-workers[16] used the low sodium diet in a group of 122 patients with congestive heart failure. In some the fluids were restricted, in others fluids were allowed as freely as desired, and in still others the total intake of liquids was forced up to 3000 cc. per day. They state: "With restricted sodium intake, restriction of water is unnecessary in treating cardiac decompensation, increases the discomfort of the patient and may prove deleterious". They found that forcing fluids had little effect upon the rate of improvement of most patients with congestive heart failure.

Wheeler, Bridges, and White[17] have recently reported the results of this regime in a group of 35 patients with congestive failure. They state that the results were excellent in 22, and that no patient was made worse by the administration of fluids in large quantities. It should be noted that the total amount of fluids administered to the patients in these two studies was considerably less than those advocated by Schemm, and it is not clear that attempts were made to provide an acid ash diet, but rather one with a neutral ash. To this extent they cannot be regarded as tests of the strict Schemm regime. I can testify from personal experience that strict adherence to Schemm's plan sometimes leads to highly satisfactory results when even slight modifications have been less successful.

There are few experiences so gratifying as to place a chronically waterlogged patient on this treatment and watch the steady improvement that follows, with the accompanying rise in the morale and courage of the patient. It would be difficult for me to express adequately my admiration for this splendid contribution, and the least I can do is to urge all of you who are not familiar with it, and who may be responsible for cardiac patients, to read Schemm's papers and give his plan the benefit of careful trial.

Coronary Atherosclerosis and Cholesterol: One of the most serious problems relating to heart disease in the adult is the appalling incidence of coronary arterial disease. It is surely unnecessary to remind you that each year thousands of apparently healthy men between the ages of 20 and 50 years are killed or reduced to invalidism by sudden and unexpected manifestations of coronary insufficiency or coronary thrombosis. For many years students have been trying to learn why changes occur in the intima of human coronary arteries that lead to gradual or sudden occlusion, why such changes affect the male so much more frequently than the female, whether there is any way of preventing or retarding these pathologic processes, and what part is played by cholesterol. It is perhaps impossible to give final answers to these questions, but progress has been made in the past few years, and some of this may be indicated briefly.

With regard to the much higher incidence in the male sex, Dock[18] has recently made observations of great significance. In studying the epicardial coronary arteries of newborn infants, he found that the intima was much thicker in males than in females, and he believes this establishes the basis for the sex difference in the incidence of coronary thrombosis. Careful measurements of the intima and the media of these arteries showed that the average intima in newborn males was 26.5 per cent the thickness of the media, while in females it was only 8.2 per cent. In Dock's words: "The male therefore begins life with about three times as much coronary intima as the female." This very important observation should be confirmed in a much larger series of infants, but there is no reason to question its essential accuracy. Dock is careful to point out that it throws light chiefly upon the sex incidence of coronary arterial disease, and states that hypertension and faulty cholesterol metabolism are the most important immediate causes.

Hypertension will be considered later; for the moment let us review

in a most inadequate way some of the present knowledge relating to cholesterol. There are many facts that point to a close relationship between this substance and coronary atherosclerosis. It is known, for example, that cholesterol in the blood serum rises to high values in those diseases which are associated with advanced coronary atherosclerosis, even in young patients; these diseases include uncontrolled diabetes, myxedema, familial xanthomatosis, chronic glomerulonephritis in young people, and some cases of obesity. It is known that cholesterol is deposited in the walls of coronary arteries, and it appears to be directly responsible for thrombosis in a large number of cases. Steiner and Domanski[19] have shown conclusively that the average level of the serum cholesterol is much higher in patients with coronary disease than in control subjects, and that it also fluctuates more widely. Coronary disease is said to be relatively rare in those races, such as the Chinese, who live on a diet low in cholesterol. Arterial lesions are readily induced in rabbits by the feeding of this substance, but this observation has been regarded as rather inconclusive because of the herbivorous nature of the experimental animal and the frequency with which it develops arterial disease spontaneously or in response to various factors. However, Steiner and Kendall[20] have recently succeeded in producing arterial lesions in dogs similar in distribution and morphologic character to those seen in human atherosclerosis and arteriosclerosis by inducing prolonged hypercholesteremia and simultaneously depressing thyroid function by means of thiouracil. It had been shown previously that prolonged elevation of the serum cholesterol alone did not produce such lesions.

It is widely believed, and frequently stated, that the amount of cholesterol in the serum of humans cannot be influenced by diet,[21] but there is apparently insufficient evidence to support this categorical statement. It is true that the level remains fairly constant in normal people, and it seems clear that it is not altered appreciably by a single large feeding of cholesterol, or even by feeding large quantities for several days. It is known that the body can synthesize this substance from foods other than those containing cholesterol. But it has been shown conclusively that the serum level can be raised by 30 to 50 per cent or more in monkeys,[22] dogs, and humans by feeding egg-yolks for days or weeks, and that the level falls to normal after such feeding has been discontinued. Steiner and Domanski[23] found that the administration of 100 Gm. of egg yolk powder per day for six to ten weeks caused an increase of

the serum cholesterol from 50 to 218 mgm. per cent, the average rise being 101 mg. per cent. Okey and Stewart[24] showed that there was a slight consistent rise in the serum cholesterol in a group of normal women fed four egg yolks daily. Kempner[25] has reported, in a lecture given at this Academy, that the average fall in cholesterol of 79 patients on a rice diet was from 243 to 189 mgm. per cent. Dr. Steiner[26] tells me that some of his patients show at least a moderate reduction in the serum concentration when placed on diets low in fats and cholesterol, and I am able to confirm this statement on the basis of personal observations upon a small group of patients. Adlersberg,[27] Steiner and Domanski,[28] and others have shown also that the level can be appreciably lowered by the administration of soya lecithin but this effect continues for only about five weeks, after which it gradually ceases, even though lecithin is continued. A consideration of this and other evidence leads me to the conclusion that the serum cholesterol in humans is not entirely independent of diet.

The observations just enumerated seem to me of probable great significance, but they cannot be regarded as wholly conclusive. There are still many and wide gaps in our knowledge of the absorption, utilization, synthesis, and deposition of cholesterol in various tissues. It seems probable that a fundamental fault in the metabolism of this substance is responsible for the high serum levels that are found in certain diseases, and there are some thoughtful students who seriously question whether a high concentration of cholesterol in the blood does *of itself* lead to coronary atherosclerosis. So far as I am aware, there is no direct proof that lowering of the serum level by means of diet or lecithin prevents further changes in the coronary arteries of those who have already suffered thrombotic closure of these vessels. Obviously it will be difficult to secure such proof. The evidence available today is indirect but strong, and to me it appears to justify the tentative conclusion that a high level of cholesterol in the blood serum is probably dangerous, and that efforts should be made to reduce it to normal.*

A moment ago I alluded to the recent report of Steiner and Kendall,[20] indicating that arterial lesions similar to those of humans have

* While the present paper was in press, further significant observations upon the probable relationship between high serum cholesterol and coronary atherosclerosis have been published by Boas, Parets, and Adlersberg (Hereditary disturbance of cholesterol metabolism: a factor in the genesis of atherosclerosis, *Am. Heart J.*, 1948, *35:* 611)

been produced for the first time in dogs by the maintenance of hypercholesteremia for some months. If they or others prove that such lesions can be produced uniformly in such animals by this means, it will provide powerful, but not absolutely conclusive, evidence that an excessive amount of cholesterol in the serum is one of the important factors in the etiology of intimal changes in the coronary arteries. It will also provide us for the first time with an experimental method for the careful and controlled study of the development of these changes, and this may well lead to the discovery of means for retarding or preventing them.

Anticoagulants in the Treatment of Cardiac Infarction: Several years ago Nichol and Page[29] reported their observations upon the use of dicoumarol in patients who had sustained coronary thrombosis. The purpose of administering this anticoagulant was to prevent extension of the original thrombus as well as the formation of new thrombi in the heart or in the systemic veins. Their experience seemed encouraging. Within a very short time similar reports, with equally gratifying results, appeared by Peters et al[30] and by Wright.[31] Dr. Wright was so impressed by the possible importance of the treatment, and by the necessity of determining its value at the earliest possible time, that he proposed a coöperative study, to be undertaken under the auspices of the American Heart Association and carried out in fifteen or more hospitals throughout the United States. It was his belief that it would be necessary to have careful and conclusive studies upon not less than a thousand, and preferably several thousand, cases and an equal number of controls. It is encouraging to be able to report that his suggestion was received with enthusiasm by the directors of the American Heart Association, and that financial support of the project was received from the U. S. Public Health Service. Under a committee representing every participating hospital, and under the able chairmanship of Dr. Irving Wright, the study has now been under way for almost a year. Under the plan adopted, all patients with acute coronary thrombosis who are admitted on odd days of the month are treated with dicoumarol; all who enter on even days of the month are kept as controls. Actual figures cannot be given today, but a preliminary analysis of the results in June 1947 showed a remarkable difference between the two groups. If such a difference is maintained throughout the study of one or two thousand cases, it seems certain that the use of dicoumarol will

be a magnificent contribution to the treatment of this serious condition.*

Penicillin for Treatment and Prevention of Subacute Bacterial Endocarditis: It is probable that very few discussions of treatment could be complete if they did not include penicillin, and the present one is no exception. Until very recent years the treatment of subacute bacterial endocarditis was generally regarded as practically hopeless, despite an occasional success from the use of one of the sulfa drugs. Loewe deserves great credit for having demonstrated the value of large doses of penicillin in this disease after the Penicillin Committee had officially declared it to be of no value. Following his reports of cures after using quantities of penicillin far larger than had previously been tried, success was achieved almost uniformly, and today it is possible to state that the great majority of patients with this infection can be cured if sufficient amounts of penicillin are administered.

But there is another use of this miraculous substance that may prove to be even more important. It has been shown[32] that transient bacteremia follows tooth extraction in as many as 70 per cent of cases, and the Streptococcus viridans is the invading organism in almost all of these. Last year Favour, Janeway, and Levine[33] reported a series of 21 patients with bacterial endocarditis, in 10 of whom there had been tooth extraction, dental procedures, or obvious tooth infection preceding the onset of the endocarditis. The probability of a causal relationship between dental procedures and subsequent infection of the heart is of such great importance that further study is urgently necessary; at the moment, it seems altogether probable that extraction of teeth and other forms of oral surgery are hazardous procedures in any patient who has rheumatic heart disease or a congenital anomaly. The American Council on Rheumatic Fever of the American Heart Association has officially recommended that all such patients receive penicillin in adequate dosage just before and for some days after extraction of teeth or any other surgical procedure within the oral cavity. It is hoped that this prophylactic measure will soon become routine and compulsory throughout the civilized world.

Prevention of Recurrent Rheumatic Fever: Brief mention should be made of a similar preventive method that is being widely applied

* Some months after the present talk was given, analysis of 1000 treated and 1000 untreated cases revealed that the mortality rate among those receiving dicoumarol was 13%, while in the untreated group it was 23%. The incidence of thrombo-embolic complications was 19% in the untreated cases but only 9% in those treated with dicoumarol. The actual number of such complications was 36 per 100 patients in the control group, and 8 per 100 in the treated group.

to rheumatic fever. It is probable that no one can speak with absolute certainty on this point, but there is a good deal of evidence in support of the belief that the routine daily administration of sulfa drugs in small doses to children who have had one attack of rheumatic fever may prevent subsequent attacks. The work of Kuttner and Reyersbach[34] at Irvington House showed conclusively that in a closed institutional population this was true. It is too soon to state positively that the drug will be equally effective in large groups of children scattered through the homes of a large city and exposed to conditions that did not exist at Irvington House. But the experience thus far seems to have been favorable, and it is quite possible that in a few years we may find a great reduction in the incidence of rheumatic heart disease because of the prevention of rheumatic recurrences by this drug.

Hypertension: We come finally to a consideration of another of the serious and baffling medical problems–hypertension. Undoubtedly many of you will consider me as incorrigibly optimistic in thinking that there has been any progress in the treatment or prevention of this condition, and you may be right in that conclusion. But there are several aspects to which your attention is invited for a moment.

The forms of treatment advocated in recent years in addition to general medical oversight and symptomatic therapy have been chiefly three in number–dorso-lumbar sympathectomy of varying extent, exemplified well by the Smithwick technique, the rice diet of Kempner, and a diet low in sodium but not restricted to rice. I shall say but little with regard to the results of sympathectomy, for two reasons. One is that Smithwick's cases have been analyzed with great care, unusual insight, and exceptional clinical judgment by Palmer[35] whose paper is commended to all of you interested in this subject. The other is that one week from today in this Academy, Dr. Hinton of this city is to analyze the results in 440 hypertensive patients subjected to this procedure. It was my privilege to hear him present the analysis as of April, 1947 before the Connecticut State Medical Society, and brief reference may be made to one feature of his work that has already been published.[36] He and his associates have now adopted a system of rules which leads to a higher rejection of cases and an operative mortality of zero up to the time of his report. These rules are based upon a classification of the degree of change in four organs, namely, the eyes, brain, heart, and kidneys. Such changes are graded from 0 to

plus 4, and the sum of all plus signs determines whether or not operation should be performed. But Hinton states frankly that no entirely satisfactory method has yet been found for the selection of patients for sympathectomy; after all objective studies have been made, the decision must still rest to some extent on clinical judgment. His analysis of a large but selected series of his patients indicates a high percentage of improvement, both subjective and objective, after periods varying from 6 to 36 months.

No personal analysis of the results obtained by Smithwick will be attempted, since this has already been done by Palmer, from whose paper several excerpts will be read:

"A major effect of dorsolumbar sympathectomy in essential hypertension results from the relative postural hypotension, pooling of the blood in dependent parts and presumed decrease in venous return. The duration of this effect varies widely, lasting from months to years, and accounts for much, if not the major part, of both the favorable effect on the disease and the considerable disability associated with the operation.

"When the favorable effect of operative intervention is critically evaluated (persistent reduction of the blood pressure to 150 systolic and 110 diastolic or below in all positions), it is found that in the present series there has been a diminishing return of favorable results the longer the patients are followed; nearly 70 per cent early in this experience, declining to 25 per cent when patients are followed three to five years or more. But this effect has been obtained twice as frequently in this series by surgical means as by a careful medical regimen and was obtained in patients with malignant hypertension whose blood pressures were unaffected by medical management.

"From this experience sympathectomy is regarded as the treatment of choice, at this time, for malignant hypertension and may be offered to patients with early or moderate vasospastic hypertension who have no clinical evidence of advanced, irreversible, diffuse arteriolar disease.

"Prescription of operation for the patient with hypertension involves evaluation of the hypertension and attendant organic changes, the social and economic status and the personality of the patient and the objective of treatment (symptomatic relief, reduction of the blood pressure or halting the progress of the disease). Final judgment may depend on balancing the present status and estimated future of the patient with induced discomfort and disability."

No reference to this subject would be complete if it did not include mention of the work and opinion of Dr. William Goldring. He advances powerful arguments against the belief that sympathectomy is an important method of treatment, although admitting that it is the best for the alleviation of certain symptoms, especially headache. His paper which appeared February 1947[37] is a scholarly, thoughtful, and very fine discussion of both the medical and surgical management of hypertension.

Of the two other methods that are now being tried, the better

known is the rice diet of Kempner, which consists of rice cooked without salt, sugar, fruits, and fruit juices. No salt is permitted. Kempner[38] states that the average patient can eat 6½ to 10 ounces of rice per day, which provides 700 to 1000 calories; the remainder of the caloric requirements must be met by the use of sugar and fruits. Despite the low protein intake, the protein equilibrium of the patients is maintained, while the chlorides in both urine and plasma decline sharply.

Of the patients in whom the non-protein-nitrogen was determined, it remained stationary or increased in 24 per cent, and declined in the other 76 per cent from an average of 53 mgm. per cent before the diet to 36 mgm. per cent after an average of 78 days on the diet. The serum cholesterol declined in 73 of 82 patients from an average of 266 mgm. per cent to an average of 183 mgm. per cent after 91 days on the diet. Of 53 whose cholesterol was 220 mgm. per cent or more, it fell in 52, and in 37 of these it reached normal levels.

Kempner's first extensive analysis considered 192 patients with hypertension on the basis of acute or chronic primary kidney disease or with "hypertensive vascular disease." Of these, 25 died after following the diet for 6 to 81 days, the average being 25 days. Of the remaining 167 patients Kempner states that there was improvement in 64 per cent and no improvement in 36 per cent. It should be noted, however, that in this analysis he does not include the 25 patients who died after an average of 25 days on the diet. Presumably these are to be regarded as failures; if they are so regarded and are included in the analysis, we find that there was improvement in 56 per cent and no improvement in 44 per cent. Stated otherwise, slightly more than half of the patients improved, and slightly less than half failed to improve.

There were 31 patients whose electrocardiograms showed inversion of T 1, and in 11 of these the T waves became upright after varying periods on the rice diet.[25] The size of the heart as measured in x-ray films decreased in 77 of 87 patients. Retinopathy decreased greatly or cleared up completely in a number of those whose blood pressure was reduced.

It would be foolish to state that the strict rice diet is valueless in the treatment of hypertension, for many patients have been demonstrably and greatly improved. But it would be equally foolish to assume that all patients will tolerate the diet, for many have refused to continue it after longer or shorter periods, stating quite frankly that they

would rather die of the disease than suffer the torments of eating rice three times a day for interminable weeks or months. Any estimate of the value of the treatment must consider at least two questions: 1. How well and how long does the average moderately sick person adhere to the strict rice diet? 2. Is the rice itself the important therapeutic agent, or is the improvement due largely or entirely to the absence of sodium? An accurate answer to the first question is not yet available, but it is certain that some patients who have taken the rice diet faithfully for months finally declare that further continuation is impossible. Whether this number is large or small is still undetermined; on the basis of my own limited experience it seems probable that a fairly large percentage lack the grim determination that is necessary to adhere to a diet that is extremely unpalatable and psychologically depressing. With respect to the second question information is beginning to accumulate; Dr. George Perera has recently reported[39] that hypertensive patients whose blood pressure had been moderately reduced by a strict rice diet could be changed to a general mixed diet very low in sodium without any rise in the blood pressure. If the rice diet was resumed, the pressure remained unchanged. In other words, the blood pressure remained at the same level if the patients used a strict rice diet or a general diet very low in sodium. Several of my patients who had been on the strict rice diet for many months have now been using a liberal diet low in sodium for periods of six to nine months. In none has there been a rise in the blood pressure or any evidence of diminution in renal function. The patients have gained in weight and strength and are far more cheerful; while on the rice diet they were extremely depressed.

It is my own present impression—and I state it only as an impression—that it is the very low sodium content of the rice diet which accounts for its effectiveness in the 50 to 60 per cent of hypertensive patients who show improvement. This impression is strengthened by the experiences of Grollman et al,[40] whose report upon five patients appeared two years ago. They used diets containing 2000 calories and less than 1 Gm. of sodium chloride. In two of the five patients the blood pressure declined to normal levels, rose again when NaCl was added to the diet, and once more fell to normal when salt was withdrawn. A third patient displayed moderate reduction of the pressure. A fourth had no reduction of pressure within several weeks. The fifth

had carcinoma of the liver in addition to hypertension; the reduction of sodium had no effect upon the blood pressure in this patient until a collapse reaction developed. There was prompt recovery following the administration of saline solution. This was the only one in whom the restriction of sodium caused any harmful effects. These observers employed low sodium diets because they had previously found such diets effective in reducing the elevated blood pressure of rats with experimental renal hypertension.

Let me conclude the inadequate discussion of this topic by expressing the personal view that we cannot as yet make any final appraisal of the three methods just mentioned. That each one has been effective in reducing blood pressure and abolishing symptoms for short or long periods in a certain percentage of patients cannot be questioned by any fair-minded person; that each one has serious shortcomings as a method of treatment must also be admitted. It is my belief and very earnest hope that all three will be abandoned within a short time, but until the specific cause or causes of human hypertension have been discovered and specific remedies found, it is of the utmost importance that low sodium diets should be given extensive and critical trials. In my opinion sympathectomy is a justifiable procedure in carefully selected patients with malignant hypertension and in a few of those whose pressures and symptoms are disabling and have not responded to any other form of therapy.

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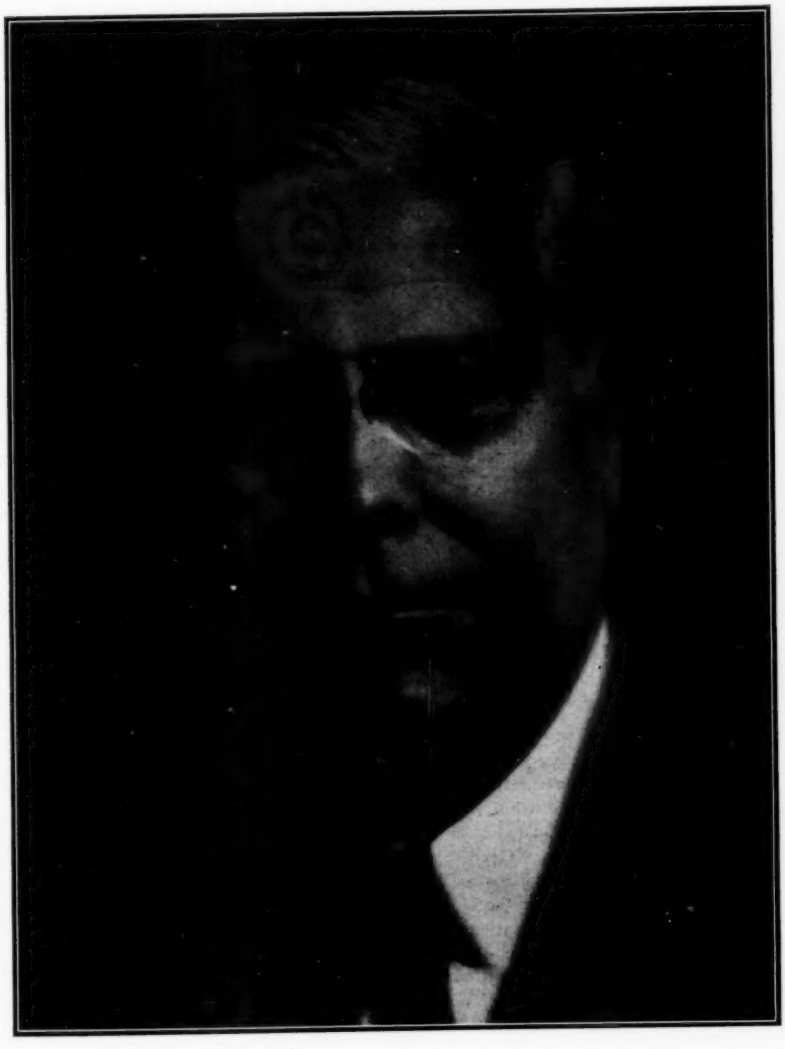

JAMES ALEXANDER MILLER

1874-1948

James Alexander Miller
1874-1948

Dr. James Alexander Miller, thirty-sixth President of The New York Academy of Medicine died on the 29th day of July 1948 in his seventy-fifth year. Thus ended one of the most distinguished careers in the history of American Medicine.

Born March 27, 1874, he graduated from Pingry School, Elizabeth, New Jersey, at the age of fifteen, and entered Princeton in the fall of 1889. Always a student, he maintained a high scholastic standing, especially in Chemistry and Latin, and yet he was in no sense a "grind." He found time to play and attained as high a degree of efficiency in the games he chose to play as he did in scholarship.

He received his A. B. in 1893 and A. M. in Chemistry in 1894, and at the age of twenty entered the employ of the New Jersey Zinc and Iron Company in Elizabeth, New Jersey as chemist. After a few months with this company Dr. William H. Park, Director of Research Laboratories of the New York City Department of Health offered him the position of Research Chemist which he accepted.

Dr. Hermann M. Biggs, who had watched his work, urged him to study medicine, and in the fall of 1895 he entered the College of Physicians and Surgeons, retaining his position in the Laboratory during his first year. In the Medical School he partly supported himself by tutoring. Graduating among the first ten men in his class, he thereby qualified to compete for the Harsen Prizes for proficiency in examinations, and won the first prize of $500. He also won first place in competitive examination for internship at Presbyterian Hospital out of a field of twenty-nine candidates.

Immediately after completing his internship, he began the practice of medicine in the summer of 1901 at Paul Smith's in the Adirondacks, and continued his practice there for eight summers. There began his long and intimate friendship with Dr. Edward Livingston Trudeau, who was largely responsible for directing his interest to the field of pulmonary diseases, especially tuberculosis. Dr. Trudeau, when asked once about Dr. Miller, replied in his droll way: "Miller?—why he is all right. He can hear everything in a chest that I can."

In the fall of 1901 he started practice in New York City, and became associated with Dr. A. A. Smith. That association continued for over ten years.

In 1903 he began his connection with Bellevue Hospital as Adjunct Assistant Visiting Physician. That connection was unbroken until his death. He was Secretary of the Medical Board from 1918 through 1927. He conceived, organized and developed the Bellevue Hospital Chest Service, the most outstanding service of its kind in the city and probably in the country. It will be a lasting memorial to him.

In World War I, at great personal sacrifice, he served in France with the American Red Cross with the rank of Major, from June 1917 to November 1918, ten months of which time he was assigned to work with Dr. Livingston Farrand as Associate Medical Director of the Rockefeller Commission for the Control of Tuberculosis in France. France honored him by making him a Chevalier of the Legion of Honor.

Dr. Miller was connected with many hospitals during his life, as Attending or Consulting Physician. He was still on call to the Post-Graduate, Woman's, Methodist of Brooklyn and the House of Rest, Sprain Ridge, Yonkers. He was also Consulting Physician to the Trudeau Sanatorium, of whose Board of Trustees he was President from 1927, declining re-election in 1945.

In spite of his multifarious and exacting professional duties he was interested in many other activities. He was a former President of the National Tuberculosis Association and the New York Tuberculosis and Health Association, in the formation of which he took a leading part. He was a member of the Board of Managers of the Community Service Society, a merger of

the Charity Organization Society and the Association for Improving the Condition of the Poor, on one or both of which organizations' governing boards he had served for over thirty years. He became an active member of the Milbank Memorial Fund in 1922, and served until January 1948. He was elected Alumni Trustee of Columbia in 1945.

Dr. Miller was a former President of the Practitioners Society, the American Climatological and Clinical Society, the American College of Physicians and the New York Medical and Surgical Society. He was also a member of the Association of American Physicians and the American Association of Thoracic Surgeons.

His term as President of the College of Physicians was a most important one. He insisted that the Annual Meeting in his presidential year should be held in New York City, the first time it had ever been held there. He worked tirelessly to make it what was generally admitted the best meeting the College had yet held. It was largely due to his enthusiasm that the College acquired its attractive and hospitable headquarters in Philadelphia. He was on the College's Board of Regents for many years, and in recognition of what he had done for it and the profession, he was made a "Master" in 1943.

His service to The New York Academy of Medicine, to which he was elected in 1904, really began in 1911 when he was appointed to the newly authorized Committee on Public Health, now Public Health Relations. He was elected its first Secretary and served as such till 1928, when he became Chairman. It is difficult to think of that Committee without associating it with Dr. Miller, for he worked tirelessly for it for over thirty seven years. He was elected President of the Academy for the years 1937 and 1938, and did much to stabilize its finances and to bring about better coöperation with associated societies. He served on the Board of Trustees from 1925 until 1943 when he declined renomination. He also served most helpfully during a number of years on the Committees on Budget, Gifts and Bequests, to Investigate Problems Related to Medical Practice, and Medicine and the Changing Order.

Dr. Miller was one whose services and attainments were bound to be recognized during his life. He was awarded the Honorary Degree of Doctor of Science by Columbia in 1930: by Princeton in 1936: and Doctor of Public Health by New York University in 1937. In 1944 the National Tuberculosis Association voted him the Trudeau Medal "for meritorious contribution in the Cause, Prevention and Treatment of Tuberculosis." A little more than a year ago, at the Convocation closing the Centennial Celebration of the Academy, he, "the Academy's most distinguished and beloved Fellow, and one of the greatest benefactors of mankind," was awarded the highest honor the Academy can bestow: the "Academy Medal." In May 1947, fifty-eight years after he had graduated, the Alumni Association of the Pingry School voted him its "Award for Winning his Letter in Life."

Dr. Miller was an extraordinarily able man. Endowed with a fine mind, he developed it to the highest degree. He was wise in counsel; discerning in judgment; unfailing in leadership, with a limitless capacity for work. When he started any thing, he never stopped until the job was completed. He gave of himself without stint. He had vision; was a straight thinker and was able to crystalize his thoughts. With it all he was intensely practical, and a wonderful judge of men. He knew how to arouse interest in a problem, and how to keep that interest. Even the politicians of the day listened to him and respected him.

He had a keen sense of kindly humor. He had a love of people and boundless kindness toward people. That is why his patients believed in him, trusted him implicitly, and never forgot him. One wrote to Mrs. Miller: "I have come to look upon him*** as a warm-spirited friend whose sympathy and understanding were a wonderful help in some dreadfully dark hours.*** Dr. Miller earned high honors during his long and busy career, and he deserved them richly; but I like to think that his greatest monument is in the hearts of all of us whom he led strongly by the hand on the long hard climb from the neighborhood of death back into the sunshine again."

His character was not one sided. While he had a great capacity for work, he also had a great capacity for play, and when he had the opportunity in his busy life, he played as he had worked, with great skill, enthusiasm and with his characteristic will to win.

"He lacked none of those attributes one would expect to find in the good Christian man that he was. He was an elder of the Brick Presbyterian Church for many years."

Granted that in this world of ours no individual is indispensible, no loss irreparable, it is difficult to believe that any who knew Jim Miller will ever cease to miss him as adviser, leader, physician and FRIEND.

MALCOLM GOODRIDGE
PHILIP VAN INGEN

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